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NEUROLOGICAL ABNORMALITIES

THEIR OCCURRENCE AND SIGNIFICANCE AS ILLUSTRATED BY AN EXAMINATION OF 500 MENTAL DEFECTIVES

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INTRODUCTION

It is now, we think, an established clinical fact that certified mental defectives display many disordered neurological phenomena other than the mental ones so symptomatic of their cerebral amentia. The problem thus constantly arises: are such defectives suffering from superadded nervous disease, or are all the clinical symptoms, mental and neurological, due to one and the same cause—an ill-developed central nervous system? It is usual in clinical descriptions of amentia to classify cases into primary and secondary amentia, the primary being supposedly due to hereditary agenesia of the nervous system, and the secondary cases due to intercurrent disease or injury. There has been, and still is, a considerable difference of opinion as to the relative proportion of cases which should be allocated to each group, and those who favour secondary factors as the principal causative influences in amentia claim that neurological abnormalities are indicative of partially recovered lesions which have been acquired during prenatal or postnatal existence. They do not, however, consider the possibility that even where there is a clear unequivocal history of trauma or inflammation such lesion may have been an incidental phenomenon occurring in a brain already imperfect, and in the *Stoke Park Monographs on Mental Deficiency and Other Problems of the Human Brain and Mind*, volume I, several cases were recorded where the patient, in addition to his mental deficiency, presented neurological phenomena which simulated the syndromes of nervous disease, but which on thorough investigation were found to be due, not to disease, but to a totally

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different cause—namely, an imperfectly developed nervous system. We were, therefore, led to suspect that the neurological phenomena presented by many defectives, as indeed by many other mentally weak but not certified patients, might really be due more to an ill-developed cerebral cortex than to disease in the cortex itself or in other part or parts of the central nervous system. We therefore decided to examine the problem, and to do so on a sufficiently extensive scale as to expect some light to be thrown upon it.

METHODS

We have for this purpose carried out a clinical neurological examination of 500 certified defectives—289 females and 211 males. The age distribution of the series ranges from 5·2 years to 41 years, with a true mean of $19{\cdot}6 \pm 0{\cdot}21$ years, and a standard deviation of $7{\cdot}2 \pm 0{\cdot}15$, which means that nearly four-fifths of the 500 cases are between 12 and 26 years of age.

For the purposes of our investigation, these 500 cases are divided, not into their legal classes of idiots, imbeciles, feeble-minded and/or moral defectives, which have no clinical or biological significance, but into two broadly differentiated groups, as follows :

Group I.—High grade aments with an intelligence quotient of 50 and upwards, but considerably less than normality. In this group there are 167 cases, of which 46 are males and the remaining 121 females.

Group II.—Low grade aments with an intelligence quotient below 50. This group comprises 333 cases, of which 165 are males and the remaining 168 females.

For every one of these 500 cases an ordinary routine neurological examination has been made, the basal features of which are graphically set forth in figs. 1, 1a, and 1b. From fig. 1 it will be seen that 19 observations have been recorded, which collectively, even though crudely, afford an indication of proprioceptive and exteroceptive sensibilities, central coordinations, effector mechanisms and other nerve phenomena usually associated with manifestations of disease. In carrying out the sensory tests pain was tested by a needle, localization by touching with a soft brush (the patient being required to point to within 2 inches of the place touched), and discrimination by means of the æsthesiometer test, errors of more than 5 cm. on the leg and 2 cm. on the back of the wrist being taken as indicative of abnormality. Postural sensibility was tested in the feet by asking the blindfolded patient to say whether his toes were pointing up or down after passive movement, and in the hands by getting him to imitate in one hand simple movements carried out by the examiner in the other hand. The latter test proved the more difficult. Bechterew's sign is elicited by percussing the extensor tendons of the back of the hand, when a flexion of the fingers is produced instead of the normal extension. In testing the knee-jerk, Strümpell's method of grading the response was employed, the increased reflex being considered

indicative of organic disturbance if it was obtainable by striking the subcutaneous surface of the tibia with the percussor.

RESULTS

In fig. 1 are set forth, in percentages, the numbers of neurological abnormalities detected in both groups. It is necessary, however, to note that these percentages are comprehensive, and do not differentiate between increased or diminished reactions, or between unilateral and bilateral manifestations. Such differences are graphically displayed in figs. 1a and 1b. The purpose of the first graph (fig. 1) is only to indicate in the clearest possible manner the departures from normality of both groups, but it proves that the lower the mental grade the greater the tendency to the manifestation of neurological abnormality. For example, fig. 1 shows that 9·1 per cent. of the higher grade cases exhibit a reduced sensibility to pain, whereas for the lower grade cases the percentage is more than doubled, being 19·5 per cent., and this tendency for the lower grade cases—that is, all those with the less well-developed mental reactions—to increased neurological departures from normality, is everywhere and strikingly obvious. As the abscissa in the graph in fig. 1 represents normality, for both mental and neurological phenomena, it is an observation of considerable significance to note that the more the mental phenomena depart from the normal, the greater is the tendency to the manifestation of neurological abnormalities usually regarded as being symptomatic of disease.

In addition to the 19 observations recorded in fig. 1 we also examined and noted other neurological abnormalities and phenomena, among the more important of which, with their percentage incidence in both groups, are the following :

	Group I. 167 cases.	Group II. 333 cases.
Nystagmoid jerking of the eyes on lateral fixation	8·6	5·4*
Coarse tremors	4·3	5·7
Tics of various types	0·5	3·0
Paresis of tongue, face or palate	1·7	7·8
Strabismus, convergent or divergent	12·5	12·0
Catatonia (slight)	3·5	7·5
Convulsions	3	13
Temper tantrums	23	26

* Fixation of the eyes could not be obtained in many low grade cases.

A point of some interest arose in connection with the group of cases showing fine choreic movements. It was found that the incidence of this

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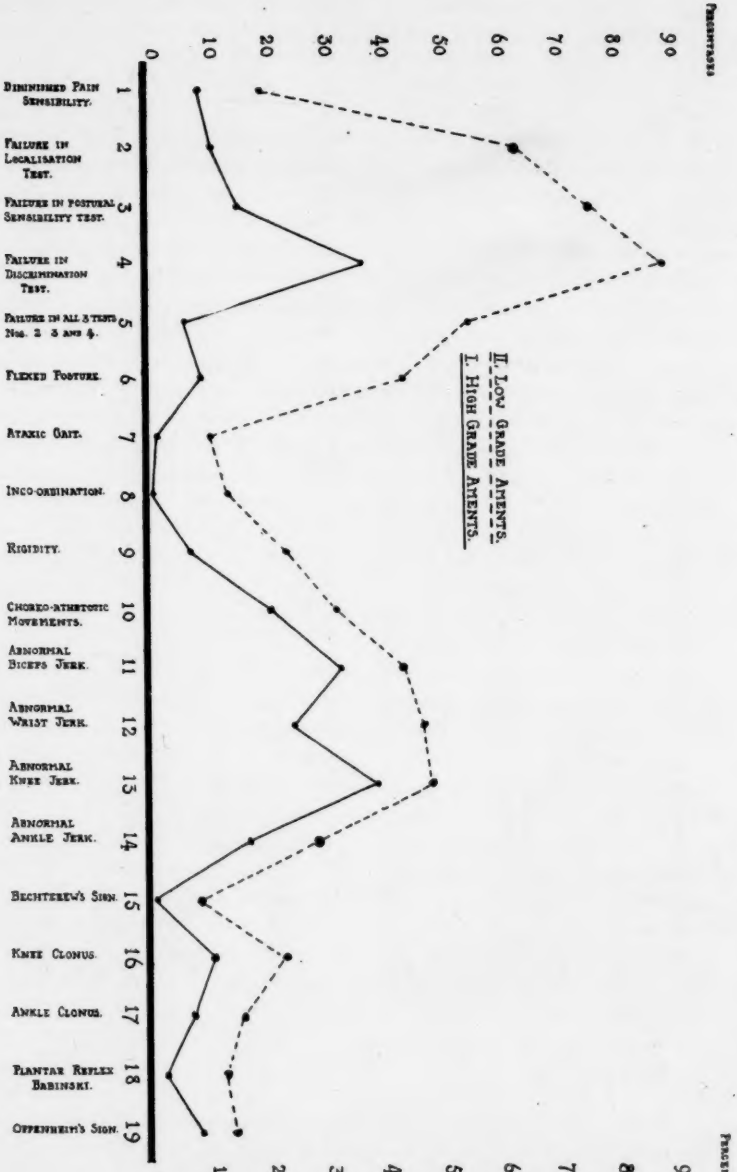
Diminished Pain Sensibility.
Failure in Localisation Test.
Failure in postural Sensibility test.
Failure in Discrimination Test.
Failure in all 3 tests Nos. 2 3 and 4.
Flexed Posture.
Ataxic Gait.
Inco-ordination.
Rigidity.
Choreo-athetoic Movements.
Abnormal Biceps Jerk.
Abnormal Wrist Jerk.
Abnormal Knee Jerk.
Abnormal Ankle Jerk.
Bechterew's Sign.
Knee Clonus.
Ankle Clonus.
Plantar Reflex Babinski.
Oppenheim's Sign.
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
0 10 20 30 40 50 60 70 80 90
Percentages
II. Low Grade Aments.
I. High Grade Aments.

Fig. 1.

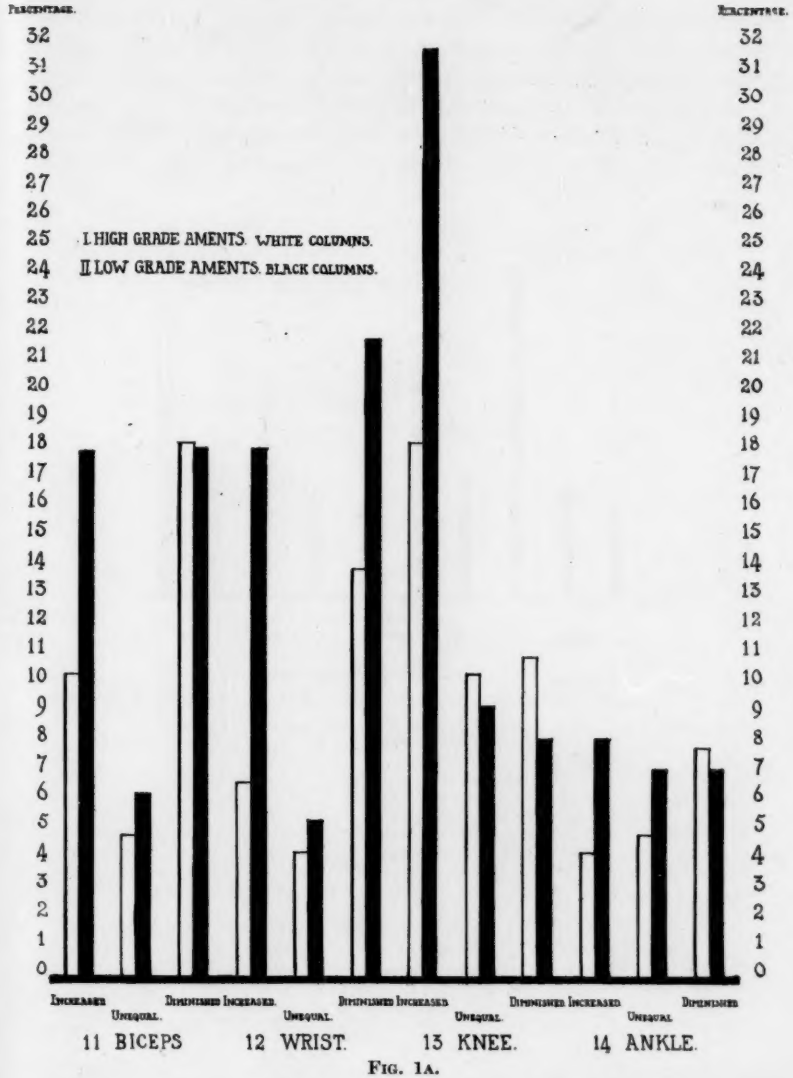

FIG. 1A.

abnormality was greatest among patients between 12 and 16 years of age, occurring in 44 per cent. of this group as compared with 31 per cent. in age-group 8–12 years and 26 per cent. in age-group 16–20 years. As Sydenham's chorea is also more prevalent at about the time of puberty, it is possible that a common infective factor is present in these two classes of patient.

DISCUSSION

From these subsidiary observations, as also from the major ones recorded in figs. 1, 1a, and 1b, there emerges the same generalized fact, that the lower the mental grade of the individual, the greater the tendency to the manifestation of certain irregular nervous phenomena which seem to simulate, but are not necessarily due to, disease. To us, as to those of our colleagues who have

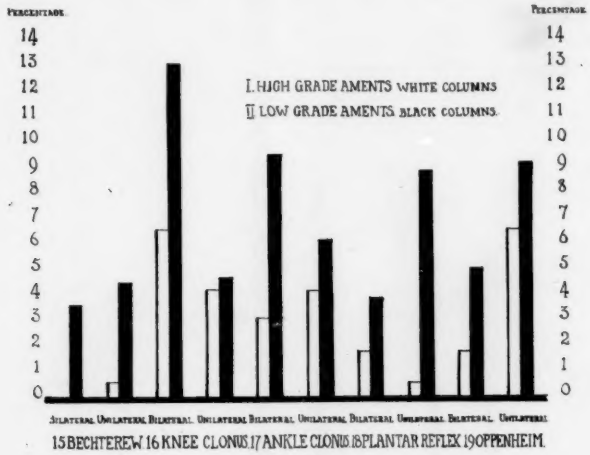

FIG. 1B.

FIG. 1.—A graph showing the percentage occurrence of neurological abnormalities in

(1) 167 high grade aments, and
(2) 333 low grade aments.

The abnormalities investigated are numbered and named below the abscissa. The percentage scales are on the right and left.

In this graph all the abnormalities are exhibited collectively, in order to show that the lower the mental grade the greater the tendency to the manifestation of neurological abnormalities. This is so pronounced, and the parallelism between the two grades is so obvious, as to suggest that the prevalence of such abnormalities can only be due to some common underlying factor other than disease, infection or injury.

In figs. 1a and 1b are shown nine of the observations recorded in fig. 1, here displayed according to their clinical occurrence as increased, unequal or diminished reflexes; or as unilateral or bilateral manifestations of certain standard neurological tests. Both these supplementary graphs again suggest the presence of some underlying factor common to both groups of patients and responsible for the manifestation of the neurological abnormalities recorded.

had these patients under close observation, both the fact and the evidence appear to be irrefutable. For the explanation there is, of course, much more room for differences of opinion, but to us it would appear that there are only two possibilities:

1. Birth injuries and/or cerebral infections and inflammations.
2. Maldevelopment of the central nervous system.

1. *Birth Injuries and/or Cerebral Infections and Inflammations.*—Given these causes there should be irrefutable evidence of difficult labour, cranial damage by forceps, or inflammatory disease (encephalitis) and in each instance such injuries or disease should be attended and followed by some fairly definite clinical conditions of paralyses—partial or complete—or other

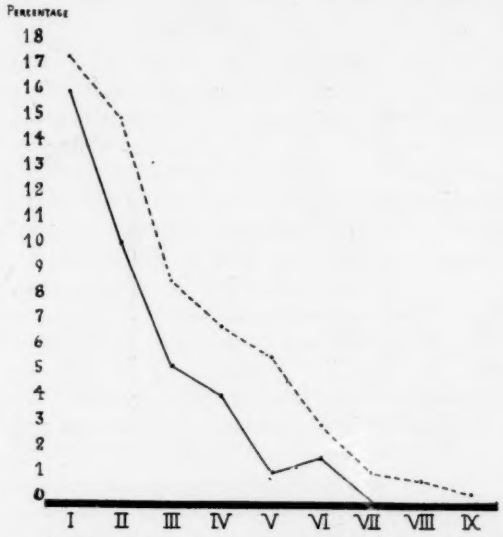

FIG. 2.—A graph showing in low and high grade aments the percentage occurrence of nine signs clinically regarded as symptomatic of pyramidal cell or tract lesions. (The abdominal reflex was found to be too variable to be useful.) The interpretation of this graph is as follows :—

Commencing on the left, it is seen that 16 per cent. of the high grade group (continuous line), and 17·5 per cent. of the low (dotted line), manifested one only of the nine clinical signs, whereas 10·2 per cent. of the former group, and 15 per cent. of the latter, individually exhibited two of such signs, and so on. The graph further shows that none of the high grade group manifested more than six of the nine clinical signs, whereas 0·3 per cent. of the low grade group reacted to all of the nine signs symptomatic, in whole or in part, of the lesion investigated.

In the manifestation by these high and low grade aments of signs symptomatic of pyramidal cell or tract lesions there is again the same parallelism between the two groups as in the other graphs, and there is precisely the same tendency for the lower grade group to exhibit an increased exuberance of neurological abnormalities. There thus again seems to be some factor other than injury or disease common to both groups and productive of the abnormalities exhibited by both.

unmistakable evidence of cerebral laceration or hæmorrhage. In these 500 cases the histories are not available, and we are therefore unable to say whether there is or is not the irrefutable evidence necessary to justify the conclusion that the mental condition was due to such causes. An examination of our records shows that only a small proportion (10 per cent.) of the 262 cases showing one or more signs associated clinically with interference of the

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pyramidal tracts could be classified as hemiplegias, paraplegias, or diplegias. That is to say, these 26 patients exhibited spasticity, contractures, and a degree of paresis, which clearly differentiated them from other defective patients. These syndromes were distributed among the groups in the following manner, the figures showing the number of cases :

Hemiplegia		*Paraplegia*		*Diplegia*	
Group I	Group II	Group I	Group II	Group I	Group II
2	8	0	1	0	15

The only other clinical syndrome found was that of postencephalitic Parkinsonism, which was clearly present in two Group II patients and doubtfully present in one Group I patient. Even assuming that the diplegias have all been produced by exogenous factors (which is denied by many investigators) it is clear that only 6 per cent. of the 500 cases showed definite evidence of disease or injury. These figures, indeed, support the conclusion that at least 90 per cent. of the amentia in our cases is of endogenous origin, and is not due to birth injuries, cerebral inflammations, or other exogenous cause. In other words, both the amentia and the superadded neurological phenomena are an exhibition of a nervous system inadequately developed in whole or in part.

Nor, in our judgment, is cranial pressure any more satisfactory as an explanation of the mental and neurological phenomena presented by the present series of cases. Physiologically, the head of every child is submitted to very considerable pressure during the natural process of a normal birth. The anatomical construction of the human skull, with its basal parts developed in cartilage, and its calvaria in membrane, is, indeed, a special device to permit of such pressure during the process of birth, and afterwards of expansion of the skull to accommodate the rapidly growing brain.

Artificial compression of the child's head and brain is, too, a widespread practice amongst many indigenous races in many parts of the world, and such compression is artificially applied for long periods of time ; yet there is no evidence whatsoever that such practices result in the production of an exogenous amentia.

Nor is the theory of the pressure of forceps or of a generalized or localized meningeal inflammation of the cerebral membranes any more logical or satisfactory as an explanation of the phenomena presented by our 500 cases.

If caused by faultily applied forceps, the injury must of necessity be localized, with localized consequences, and must equally naturally vary in the sites of its cortical application. Hence such a locally-acting causation cannot possibly be responsible, at one and the same time, for a widespread amentia and a multiplicity of neurological aberrations and reactions. Cerebral inflammations, whether of the brain or its membranes, occurring at birth sufficiently diffuse to account for the symptoms, would most probably be

followed by the death of the child, and are an even more unsatisfactory explanation.

It thus appears to us that the evidence against birth injuries, trauma and/or cerebral inflammations, as a common causative factor in these 500 cases, with their universal amentia and/or superadded neurological aberrations, is so overwhelming that we cannot accept it, except possibly in a very small percentage of cases. We are, therefore, forced to consider the possibility of the only other alternative—a maldevelopment of the central nervous system, and though we find this is a more satisfying explanation of the phenomena presented, and one more in accord with modern neurological thought, it would be idle to deny that this too confronts us with difficulties entirely its own.

2. *Maldevelopment of the Central Nervous System.*—Because a mental defective exhibits on examination neurological abnormalities in addition to his amentia, there is no reason for supposing that these added signs are due to the intrusion of an exogenous factor. All that can logically be concluded is that the nervous systems of these patients are in an unbalanced state, or rather that the nervous forces have struck a balance in a manner different from that which occurs in the normal individual. The fact that increased reflexes, for example, are frequently found should be interpreted not as evidence of disease but as proof of at least a diminished inhibition. Such abnormal signs may obviously be produced as effectively by an agenesia as by lesions, and it has been shown in the preceding discussion that it is improbable that such exogenous factors as injury or infection play anything but a small part in the etiology of mental defect. On the other hand, while it is difficult to maintain the theory of widespread or scattered lesions to account for both the amentia and the neurological abnormalities present in any one case, the very diversity of symptoms is a point in favour of the view that maldevelopment of the nervous system is the underlying common cause. For according to this view aments with neurological abnormalities suffer from an agenesia of those parts of the nervous system regulating, for example, movement and tone, while a more generalized developmental retardation of cerebral growth explains the amentia they share with their fellows.

The varying degree of amentia shown by the whole group may not improbably be explained by a defective development of the association areas. In any case Flechsig's studies on the myelinization of cortical axons have proved that all those which form the bulk of the cerebral association areas are always the last to myelinate. Subsequent work also supports the view that it is precisely these association areas which, in the feebleminded, fail to attain a full development. Assuming such a partial failure of parietal association areas—of which we have in our possession both macroscopic and microscopic evidence—there is afforded an adequate and sufficient explanation of the comparative failure of these defectives in the sensory tests to which they were submitted.

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As regards other portions of the nervous system, the developmental errors of these find their counterpart in the neurological abnormalities described in this paper. It is obviously impossible to localize the errors producing these abnormalities more accurately than to indicate the systems—receptor, internuncial, or effector—which may be involved. For example, the fine choreic jerking shown by many patients may theoretically be produced by interference with, or diminution of, any of the numerous streams of impulses passing through receptor and cortical internuncial neuronic elements to the principal motor neurones whether Rolandic or striate. Again, it is even unwise to state that the presence of the signs associated in disease with lesions of the pyramidal tracts indicates in the mental defective a diminution or destruction of Betz neurones only, for it is possible that the inhibitory influence conveyed by the latter may originate elsewhere in the cerebral cortex, for example in the medium pyramidal cells of the frontal cortex. Certainly von Economo's recent studies on cerebral cytoarchitectonics support the view that a large part of the frontal area is definitely associated with the motor impulses which finally become exteriorized from the Rolandic motor area. However this may be, the majority of defectives showing increased reflexes exhibit no demonstrable spasticity or loss of power.

This difficulty in localization of the defect is especially apparent when diminution of the tendon reflexes is considered. Thus the spinal synapses themselves may be at fault, or the defect may lie on the receptor side of the arcs. Nor can a cerebellar component be excluded.

Many of the neurological abnormalities shown by mental defectives may be classified as ' release phenomena '—that is to say, they are produced by the overactivity of certain parts of the nervous system which have been released from an inhibitory influence normally present. In the ament this defective control may well be due to the maldevelopment of the phylogenetically most recent part of the nervous system, that is, the cerebral cortex, and in particular its supragranular portion. On the receptor side this lack of control is illustrated in those patients having reduced pain sensibility. If, in these patients, the intensity of stimulation surmounted their raised pain-threshold a violent ' explosive ' withdrawal reaction was often obtained. This primitive non-discriminative response is characteristic of individuals in whom the thalamus has been partially isolated from the cerebral cortex.

On the effector side the increased tendon reflexes indicate a diminished cortical inhibition over the appropriate spinal segments, and the same interpretation may be given to the presence of Bechterew's, Oppenheim's, and Babinski's signs. The frequency of convulsions and epileptic equivalents, such as temper tantrums, is similarly indicative of defective inhibition. The flexed posture observed particularly among the low grade patients may also be considered as a release phenomenon, for analogous attitudes are produced by a variety of conditions having in common diminished cortical activity. Furthermore, generalized flexion accompanied by marked rigidity charac-

terizes disease of the neostriatum, and it is possible that the flexion present in aments, which is usually unaccompanied by rigidity, may be due to defect of the infragranular cortex which in the process of evolution may have taken over some of the functions of the putamen and the caudate nucleus. It is well established that in low grade aments the infragranular, as well as the supragranular, cortex is often reduced in volume, and it is not difficult to conceive that the resulting reduction in the cortical activity may permit the appearance of a mild degree of generalized flexion owing to the overactivity of other nervous elements previously held in check.

Thus it appears that on neurological grounds there is much to favour the view that most of the nervous abnormalities shown by the mental defective have their origin in a maldevelopment of the central nervous system. If this be so, as we believe from the evidence before us, the observation should have an important bearing on the manifestations of abnormal neurological signs not associated with the medicolegal categories of mental deficiency. Such individuals presenting such signs in the absence of 'disease' syndromes may have within them the seeds of a hereditary cortical agenesia and may thus be predisposed to defects of the nervous system easily mistaken for disease.

CONCLUSIONS

1. Mental defectives of all grades are subject to abnormalities in the nervous system.

2. The abnormalities are due with few exceptions to a failure of the nervous system to attain a development adequate to the performance of its intricate functions.

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PAPILLOMA CHOROIDEUM WITH DIFFUSE CENTRAL NERVOUS SYSTEM METASTASES

By

GEORGE W. HALL AND THOMAS L. FENTRESS, CHICAGO

PAPILLOMA choroideum is one of the relatively uncommon intracranial tumours. Davis and Cushing [1] in 1925 reported six cases out of a series of 964, or 0·6 per cent. In 1930, Van Wagenen [2] made an exhaustive review of the literature, in which he found only 45 cases, and added two of his own. Since then, four new cases have been reported, two by Vincent [3] and one each by Spota [4] and Guillain.[5]

Although this tumour is typically localized, Van Wagenen studied the question of seeding and found that in six cases this had occurred. The first case of the kind was reported in 1868 by Le Blanc.[6] His case was a papilloma of the left lateral ventricle which metastasized to the right parietal region. Additional examples of seeding were reported by Bielschowsky and Unger [7] in 1902, Van Bouwdijk [8] in 1914, Toppisch [9] in 1925, and Gromelski [10] in 1926. The second case of Van Wagenen was also an example of seeding, in that a papilloma of the left lateral ventricle was found to have spread to the third and right lateral ventricles.

The case which we are reporting is of interest both because of the rarity of this type of tumour, and because of the clinical difficulties which were present.

REPORT OF A CASE

History.—J. R., a white male, age 61, was admitted to the service of the senior author at St. Luke's Hospital on January 3, 1932. He gave a history of having begun to feel 'run down' about a year previously. This had become progressively worse, and in the course of a year he had lost 30 lb. Eight months before admission he had girdle pains around his waist associated with mild nausea. Two months later he developed an intermittent, right sciatic pain which after three months became bilateral. There had been some vague urinary symptoms for which a prostatectomy had been done four months before admission. The past history was unimportant, except for a Neisserian infection as a young man, and a penile sore in 1898. He stated that he had never received any antiluetic treatment, and that a blood Wassermann had been negative on several occasions.

Examination.—The general examination was negative except for marked emaciation, a blood pressure of 150/104, and the recent prostatectomy scar. Neurological examination revealed that the right pupil was slightly larger than the left, but both pupils reacted normally to light and on accommodation. The ocular fundi were normal, except for slight arteriosclerotic changes. There was a right sixth nerve paresis with diplopia which was said to have been present for years. The cranial

nerves were otherwise normal. A generalized weakness of the musculature was observed without any localized atrophy or fibrillary twitchings. The tendon reflexes of the upper extremities were normal, as were the abdominal reflexes. The knee-jerks could not be obtained except by reinforcement, and the Achilles-jerks were both absent. There was a negative Hoffman and Babinski sign on both sides. There was no ataxia. The vibratory sense at both ankles was moderately decreased, although

FIG. 1.—Cauda equina showing tumour mass on left fifth sacral root.

the joint sense was normal. There was a definite diminution of response to pain, heat and cold on both legs below the knees, but no impairment of sensation in the saddle area. The Lasègue sign was present bilaterally. The urological consultant stated that the prostatic bed was normal, and a cystoscopy revealed a basal cystitis, which it was thought explained the patient's leucocytosis.

Laboratory Examination.—The blood showed a white blood count of 21,400. The reds numbered 4,620,000, and the hæmoglobin was 87 per cent. Differential count:

87 per cent. polymorphonuclears. Urine : 30 mg. of albumen and 508 white cells per high power field. B. proteus was reported in urine cultures. Gastric analysis : free acid 41, combined 21—total 62. Blood chemistry : urea 14·4, non-protein nitrogen 34·9, sugar 120.

X-rays of the chest showed a circumscribed area in the left upper lobe with striations into the surrounding parenchyma. This was interpreted as a possible metastasis. X-rays of the lumbar spine and sacrum revealed a moderate hypertrophic arthritis. Lumbar puncture on January 1, 1932, showed a partial subarachnoid block.

FIG. 2.—Tumour in pia-arachnoid covering pons. Paraffin material. Van Gieson stain. Leitz obj. 4 mm. ; ocular 10×.

The initial pressure was 250 mm. The fluid was slightly xanthochromic and contained seven cells per c.mm. Globulin 3 plus ; Wassermann negative and Lange negative. A second puncture on January 4 gave the same findings except that the pressure was 350 mm. and there were only two cells.

Course.—On admission, the patient was mentally confused and could not be certain about the date. His mental state rapidly became worse and his refusal of food and fluids made proctoclysis necessary. On the fourth day he developed an incontinence. On the eighth day his temperature rose to 101° F. Definite signs of bronchopneumonia were detected. On the morning of the ninth day he died.

Clinical Diagnosis.—Before autopsy the clinical diagnosis was tumour of the cauda equina, perhaps metastatic.

Necropsy.—This was performed by Dr. Graham Kernwein on January 11, 1932. The chief findings were a terminal bronchopneumonia, with no evidence of metastatic malignancy in the lung, as had been suggested by the X-ray report, and a small adenoma of the right suprarenal gland. Examination of the cauda equina revealed a tumour mass the size of a large olive, adherent to the left fifth sacral root just inside the dura (fig. 1). On close inspection there was an opaque, whitish thickening of the leptomeninges which was most marked over the lumbar cord and the cerebellum.

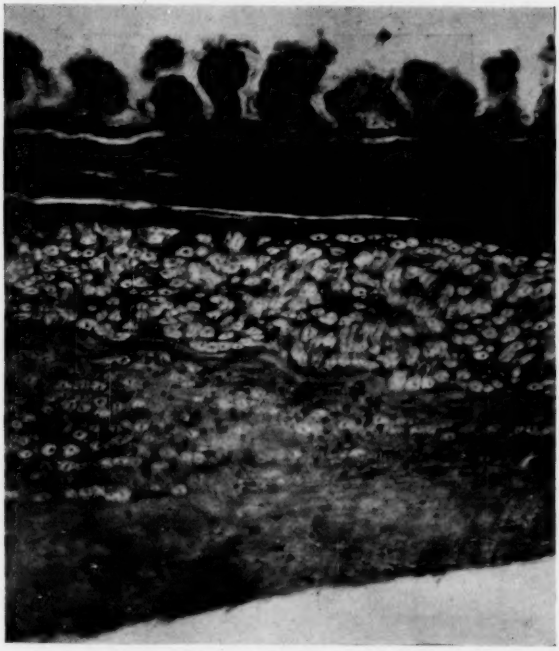

FIG. 3.—Section through wall of cauda tumour showing lining of papilloma cells. Fifth sacral root has been cut across. Hæmatoxylin and eosin.

Gross examination of the brain showed the ventricles to be of normal size and no changes of the choroid plexuses were evident.

Microscopic examination was made and sections stained with Van Gieson's method showed that the structure of the tumour was typical of papilloma choroideum. The cells could be seen to be arranged with the characteristic relations to the core of vessels and connective tissue stroma (fig. 2). Blepharoplasten could not be demonstrated. Sections through the cauda tumour revealed that it was essentially a sac, lined with papilloma cells and filled with coagulated fluid (fig. 3). The tumour cells were found throughout the leptomeninges of the cord and brain, even to the frontal pole, as well as in all the ventricles. In one section it was possible to demonstrate

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groups of tumour cells within the cerebellar cortex (fig. 4). Further examination of the brain and cord gave no evidence of central nervous system syphilis.

COMMENT

Pathological.—From a pathological standpoint, the chief problem is that gross examination of the choroid plexus of the ventricular system failed to reveal a tumour mass. In many sections, as evident in the illustration from the inferior horn of the lateral ventricle, normal choroid plexus was seen immediately beside the papilloma (fig. 5). Thus it is difficult to decide upon

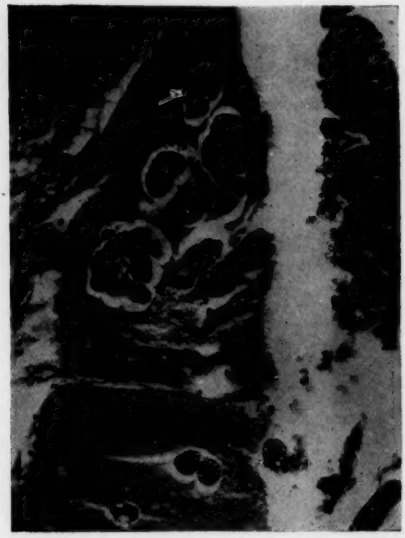

FIG. 4.—Tumour cells in cerebellar cortex. Hæmatoxylin and eosin.

the site of the primary tumour. Probably this question is best solved by the assumption that there was a generalized degeneration of the choroid plexus to form the papilloma. A section from the third ventricle reveals every gradation from normal choroid plexus to typical papilloma cells (fig. 6).

Clinical.—This case presented a most interesting clinical problem. The history of bilateral sciatic pain with bladder disturbance, the diminution of patellar reflexes, the absence of Achilles-jerks and hypæsthesia of the lower extremities, all suggested tumour of the cauda equina. The clinical findings were confirmed by lumbar puncture. However, the loss of 30 lb. in weight, and the history of general weakness before the onset of the pains, brought up the question of malignancy with metastasis. The X-ray findings of possible pulmonary metastasis (which, however, were not confirmed at post-

mortem) tended to corroborate this suspicion. The chief objection to the diagnosis of metastasis was that no clue to the location of the primary tumour could be found. The old luetic history was also a disturbing factor, despite the negative serology.

FIG. 5.—Inferior horn of lateral ventricle. Paraffin material. Van Gieson stain. Leitz objective 10 ×; ocular 10 ×.

SUMMARY AND CONCLUSIONS

1. A clinical and pathological report of a case of papilloma choroideum with diffuse central nervous system metastases is presented.

2. The phenomenon of seeding of the meninges by a benign intracranial tumour is demonstrated.

3. The difficulty of clinical diagnosis in such a case is self-evident.

4. One of the chief reasons why the possibility of the primary tumour

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being intracranial was not considered is, that metastasis from these tumours is thought to be rare.

We wish to acknowledge our indebtedness to Dr. J. Kernohan of Rochester, Minnesota, and to Dr. A. Weil of Chicago for their assistance in the classification of this tumour, and to Dr. George Funck, who referred the case to us for examination and diagnosis.

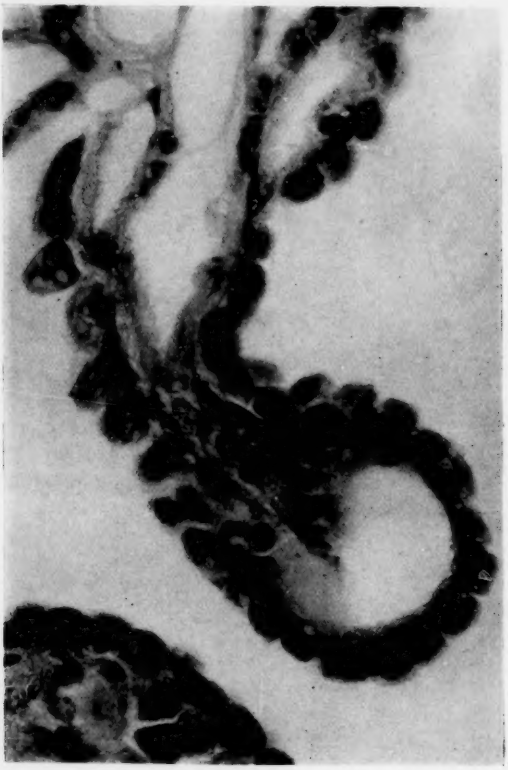

FIG. 6.—Choroid plexus of third ventricle. Paraffin material. Van Gieson stain. Leitz obj. 4 mm.; ocular 10 ×. Extension 101 cm.

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XUM

THE PROPRIETY OF DIAGNOSTIC LUMBAR PUNCTURE IN INTRACRANIAL HYPERTENSION*

By

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INTRODUCTION

In recent years there has been an increasing prejudice against lumbar puncture, from both the profession and the laity; neurosurgeons, especially, have protested against this procedure in cases showing evidence of increased cerebral pressure and in expansive posterior fossa lesions, and have asserted that there is danger of herniation of posterior fossa contents into the foramen magnum with medullary pressure and serious or fatal outcome. To quote Cushing [1] in this connection as far back as 1909: 'The withdrawal of cerebrospinal fluid from the spinal meninges by a lumbar puncture is often hazardous, as it may tend to a sudden wedging of the bulb in the opening, with anæmia and paralysis of the vital centres.' This statement has not been proved by factual evidence; it by no means follows that the presence of constricted cerebellar tonsils in the foramen magnum, found either operatively or at necropsy following lumbar puncture, is directly due to lumbar puncture, and much less follows that such a condition is due to a *sudden* wedging of the posterior fossa contents. Moreover, this cerebellar annulation is found in cases not punctured. It is rather here contended that such anatomical findings attributed to puncture are due to brain œdema, and when present take place slowly and not suddenly. No experimental evidence of movement of posterior fossa contents by fluid pressure variations between communicating ventricles, cisterna, and subarachnoid spinal spaces is reported, even in distortions of the posterior fossa. The diagnostic importance of cerebrospinal fluid analysis is unquestioned and often of deciding influence when operative intervention is considered. It is the purpose of this paper to bring forth evidence that a spinal tap performed according to a strictly defined technique is a reasonably safe and justifiable procedure in cases of intracranial hypertension. The alternative of ventricular puncture, a major operative procedure, entailing as it does skull trephine and brain puncture, is not to be lightly considered; moreover, the mortality of ventricular puncture is admittedly higher than any statistics following lumbar puncture. I should like to

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emphasize that a neurological complication following puncture is not necessarily due to puncture, and shall quote examples in confirmation thereof.

In the early part of 1932 I proposed to Dr. Jules H. Masserman, then Senior Resident in Neuropsychiatry at the Stanford Medical School, that he should undertake experiments directed, firstly, to the determination of the presence or absence of posterior fossa movement in fluid pressure variations, and secondly, the explanation of the so-called herniation of the cerebellar tonsils in the foramen magnum, in order to clear up conflicting experience and controversial points. These detailed experiments, largely due to the original ideas and painstaking labour of Dr. Masserman, have formed the subject of two papers recently submitted jointly by us to the *Archives of Neurology and Psychiatry* and soon to be published. Briefly, these experiments were as follows.

EXPERIMENTAL DATA

I. Anatomic Investigations.—These were made—

(*a*) To obtain roentgenograms of the human ventriculo-subarachnoid system under various degrees of fluid pressure.

(*b*) To investigate the effects of rapid fluid drainage of one portion of the system on the manometric pressures in other portions.

(*c*) To study the effects of artificially produced distortions of the brain on intracranial hydrodynamics.

Fresh cadavers were prepared as follows: The cranial circulation was ligated, and its pressure hydrostatically maintained. Needles were inserted into the lateral ventricles, basal cistern, and lumbar spine, and each needle was connected to a manometer. The cerebrospinal fluid was then replaced by a radiopaque solution, and control roentgenograms of the skull taken to show the outlines of the ventriculo-subarachnoid system under various degrees of fluid pressure. At each pressure level lumbar and cisternal drainages were performed, and possible movements of the intracranial contents studied by comparing subsequent encephalograms with the control. During each drainage, also, the manometric readings in other portions of the ventriculo-subarachnoid system were recorded, and the descending pressure curves plotted. Various parts of the brain were then distorted in situ by the injection of melted parowax, and the effects of such distortions on intracranial hydrodynamics studied by the above methods. The conclusions were as follows:

1. The lateral ventricles and the cisternal and lumbar subarachnoid spaces normally remained in free communication under all experimental pressure variations.

2. Rapid cisternal or lumbar release of high intracranial pressures did not produce movements of the tentorium, cerebellum, or medulla.

3. Distortion of a cerebellar lobe forced it down against the medulla and caused a partial hydrodynamic block between the lateral ventricles and the lumbar sac.

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II. The Influence of Rapid Reduction of Intracranial Pressure on the Occurrence of Brain Œdema.—This investigation is an attempt to evaluate the possible pathophysiological effects of rapid fluid decompression of the ventriculo-subarachnoid spaces.

A. Pairs of dogs were similarly anæsthetized and their intracranial pressures slowly raised by hydrostatic means from normal to 750 mm. of water. The intracranial tension of dog (A) was kept at this level for four hours, whereas that of dog (B) was rapidly reduced after the first half-hour. At the end of the four-hour period sections were taken from each brain for study. The brains were then removed and desiccated. The series of (B) brains showed a more pronounced microscopic tissue œdema and a greater percentage of water loss by desiccation than did the corresponding series of (A) brains.

B. In nearly all cases the spinal fluid pressure returns to its original level within from three-quarters to three hours following lumbar drainage of 10–40 c.c. of fluid. The rise may then continue to give a cranial hypertension lasting a variable length of time.

If more than 35 c.c. of spinal fluid is rapidly removed and the pressure is then allowed to restore itself, a second drainage of the same amount of fluid will produce a greater drop of spinal fluid pressure than did the initial drainage. This may be interpreted to indicate an encroachment on the volume of the subarachnoid spaces, either by vascular engorgement or brain œdema.

CLINICAL DATA

One hundred and three unselected cases of lumbar puncture in cases of intracranial pressure or in cases of suspected intracranial pressure are here reported, including those showing some degree of papillœdema. These latter, 12 in number, may or may not be relevant to the series, but have been included because of the considerable importance of this symptom in the cranial conditions under discussion. The following tabulations from the detailed charted records of these cases will present in concise form the pathological states and sequelæ :—

LUMBAR PUNCTURES

	Number.	Percentage.
Total Cases	103	100
Verified tumours	58	56
Diagnosed tumours, unverified ...	28	27
Brain abscesses, verified	5	5
Papillœdema	12	12
Verified Tumours	58	100
Supratentorial	39	67
Midbrain	2	3
Subtentorial	17	29
Cerebellopontile angle	6	10

	Number.	Percentage.
VERIFIED TUMOURS—*Continued*		
Choked disc, measured	16	28
Papillœdema	37	64
Spinal fluid pressure increased ...	23	40
Pressure normal	18	31
Pressure not noted	16	29
SEQUELÆ		
Incidence of symptoms (all)	11	11
Severe headache	4	4
Nausea and emesis	2	2
Convulsions	1	1
Death	4	4
None	92	89

DEATHS FOLLOWING LUMBAR PUNCTURE

Case I.—425, female child, age 4½ years. Fundi showed papillœdema. Spinal fluid pressure not noted; 4 c.c. withdrawn. No initial symptomatology or shock. Death in 13½ hours, preceded for half an hour by convulsions and bulbar failure. *Necropsy.*—Rupture of highly vascular sarcoma in left anterior thalamus. 'Cerebellum, pons, and medulla normal.'

Case II.—392, female child, age 8 years. Fundi showed choked discs. Spinal fluid pressure greatly increased, 2 c.c. withdrawn. Condition before puncture grave, cyanosis and cardiac complications, headache. Death in six hours. *Necropsy.*—Small orange-sized cystic tumour mass, right parietal region; base of brain normal.

Case III.—88,593, male, age 36 years. Diagnosis: Tuberculous meningitis. No mention of fundus examination. No definite neurological findings. Maniacal. Lumbar puncture done, 10 c.c. withdrawn, 40 mm. pressure. Became more maniacal. Second puncture two days later, 30 c.c. withdrawn, pressure not noted. Died 17 hours later. *Necropsy.*—Syphiloma of brain, floor of third ventricle. No mention of posterior fossa herniation.

Case IV.—69,649, male, age 50. Diagnosis: Cerebrospinal syphilis; one positive spinal fluid. Fundi normal. First lumbar puncture half an hour after 0·6 gm. arsphenamine intravenously; pressure 17·5 cm., 25 c.c. withdrawn. Second puncture 5½ hours later, pressure 17·5 cm., amount not noted. Third puncture half an hour after 0·6 gm. arsphenamine 16 days later, pressure 8·5 cm., 20 c.c. withdrawn. Fourth puncture 5½ hours later, pressure 10·5 cm., 20 c.c. withdrawn, 'considerable reaction.' Fifth puncture five days later, pressure not noted, 30 c.c. fluid withdrawn, replaced by mercurialized serum. Sixth puncture four hours later, pressure 18 cm., 18 c.c. withdrawn. 'Marked systemic reaction.' Three hours later complained of severe headache, soon had to be given artificial respiration. Died 40 hours after puncture. *Necropsy.*—Cystic glioma, right hemisphere of cerebellum, 5 cm.

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in diameter. 'On lower surface of the cerebellum there are cone-shaped projections on each side of the medulla, the one on the right is longer and measures about 2½ cm. in length and projects about 2 cm. from cerebellum. Corresponding process on the left measures 1½ by 1 cm.'

DEATHS IN CASES WHERE PUNCTURE WAS POSTPONED

Case I.—584, single male adult. Symptoms: dystonia, occipital pain and rigidity. Impression: tuberculous meningitis. Course in hospital: died the morning after day of entry. *Necropsy.*—Tumour 5 × 3 cm. in right cerebellar hemisphere.

Case II.—3,123, single female, age 14 years. Symptoms: sudden onset of occipital headache, left-sided weakness, right third nerve palsy. Course in hospital: became comatose, died 5½ hours after examination. *Necropsy.*—Hæmorrhage into right lateral ventricle.

DISCUSSION

Of the four deaths following lumbar puncture, only one occurred in a posterior fossa lesion, a pathological locus supposed to be particularly favourable to puncture accidents. This death occurred in one of the two cases which fulfilled the foregoing experimental postulates of improper lumbar decompression and drainage producing vasomotor shock and brain œdema; therefore these two cases cannot be reasonably included in a series of technically proper lumbar punctures and have been retained in the series because of the inclusion of every case of suspected expanding lesion. The remaining two cases of deaths both showed supratentorial lesions and the puncture may not have influenced the fatalities. That this point of view is not unreasonable is indicated by the two cases reported which died before a contemplated puncture; had a puncture been done in either of these cases it would doubtless have been incriminated with the fatality, as were the two punctured cases on the 'post hoc' premise. In none of the fatal punctured cases was death sudden, as would be expected by sudden wedging of posterior fossa contents into the foramen magnum with paralysis; the shortest time interval was six hours (P 392).

Acusticus tumour is considered to favour untoward sequelæ from puncture by dislocation of posterior fossa contents. Six such tumours were punctured in the series with no alarming after-effects or fatalities.

A number of the cases showed remarkably high intracranial pressures (P 1,229, 90·0 cm. (of H_2O); P 2,407, 85·0 cm.; P 4,266, 72·0 cm.; 58,145, 60·0 cm.), indicating communication between the cerebral ventriculosubarachnoid and spinal spaces, and the absence of a foraminal block, as has been imagined in the presence of such degrees of pressure.

The literature on the subject shows a surprisingly small incidence of immediate death following lumbar puncture as compared with the total

fatalities, and indicates that if lumbar puncture plays a rôle it is very doubtfully concerned with sudden wedging of the bulb, which would produce alarming or fatal results. Thus, in a 20-year period, 1895–1915, Schönbeck [2] in a comprehensive article practically covering the lumbar puncture period up to the time of his publication reported 71 fatalities, amongst which 37 were in tumours (11 infratentorial) and 13 due to hæmorrhage. In 10 cases death occurred after such an interval after puncture as to render the relationship questionable ; in seven cases of immediate death the majority were associated with hæmorrhage. In one internal case (Curschmann) a tuberculoma was found pressing on the floor of the fourth ventricle. In only three cases was wedging of the cerebellum reported in the posterior fossa.

An important article by Masson [3] reports punctures in 94 cases of verified intracranial tumours (32 infratentorial) without a fatality, and but one death (22-hour interval) in an unverified tumour series of 106 cases.

The fatalities of Schönbeck in the series here reported reveal a surprising predominance of supratentorial over infratentorial fatalities. In 40 punctured cases of verified brain tumour reported by Comfort [4] the only fatality possibly related to puncture occurred in rupture and hæmorrhage of a frontal tumour. Fremont-Smith and Putnam [5] recently discussed the present status of lumbar puncture as performed in the Boston City Hospital and stressed the small number of reported fatalities in recent years. They call attention to the value of very refined methods of diagnosis in early cases. In the discussion Horrax agreed that the danger was very slight with a low grade of choking. Puusepp reports 18 infratentorial tumours punctured without accident.

The considerable difference in the fatal statistics of the series of Schönbeck and of Masson represents in the Schönbeck series many cases punctured without the precautions and refinements of diagnostic technique that have been gained by experience and also many cases of actual decompression, and in the Masson series a purely diagnostic procedure carefully and intelligently done. This leads to the consideration of the technique of a properly performed lumbar puncture.

The patient should lie in a horizontal side position and the needle should not be of larger bore than 19 gauge. A mechanical manometer, as that of Claude, is preferable to a calibrated tube because of better fluid manometric control. At the first pressure reading jugular compression should be made to determine the patency of the craniospinal pathways ; should the fluid pressure not vary by compression or release, indicating a craniospinal block, fluid should not be withdrawn ; also, particular care should be observed when the fluid is xanthochromic, which may indicate a soft and vascular ventricular or surface tumour and danger of hæmorrhage. If the fluid pressure responds to jugular compression this test should be repeated after 2·5 c.c. are withdrawn, and the normal pressure, 15·0 cm., should not be reduced to lower than 10·0 cm. and an abnormally high pressure by not more than 50 per cent. No more than 5 c.c. of fluid should be withdrawn in tumour suspects, and less if any

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untoward symptoms develop, such as pain, respiratory, or pulse variations. ***Fluid withdrawal should be done slowly, drop by drop.*** After the puncture the patient's body should be inclined so that the head depends and kept so for twenty-four hours.

A satisfactory fluid analysis may be made with 5 c.c. of fluid. To quote Dr. H. A. Wyckoff, Director of the Stanford Clinical Laboratory :

' The minimum quantity of cerebrospinal fluid which will suffice for a fairly complete diagnostic examination is 4·8 c.c., used as follows :

' Wassermann—2 c.c. (qualitative).

' Colloidal gold—0·1 c.c.

' Protein, total—1·0 c.c. (quantitative).

' Globulin—0·7 c.c. (Nonne and Noguchi).

' Sugar—1·0 c.c. (quantitative), making a total of 4·8 c.c.

' This amount requires the utmost care in pipetting and does not allow for repeating any of the tests, which is sometimes desirable. There are still other reasons why it is very undesirable to have such small quantities of fluid with which to work. I earnestly advise that at least twice this amount of cerebrospinal fluid (say 10 c.c.) be sent whenever the nature of the case permits.'

SUMMARY AND CONCLUSIONS

1. Clinical and experimental evidence is here presented to support the contention that there is no proof of sudden wedging or movement of the posterior fossa contents into the foramen magnum following spinal tap in open craniospinal pathways, even in the presence of increased intracranial pressure. There is, however, reason to believe that so-called herniation is an expression of general brain swelling and œdema and may be due to changes in intracranial pressure and vasomotor paralysis and shock from various causes. Sudden and excessive fluid withdrawal by spinal tap may produce such œdema, as well as similar withdrawal of fluid from other parts of the cerebrospinal fluid system.

2. Of the four deaths in the series two can be excluded as due to properly performed diagnostic punctures ; two may or may not have been determined by diagnostic punctures.

3. The incidence of post-puncture accidents is greater in supratentorial than infratentorial tumours and the incidence of sudden death is greatest in vascular lesions.

4. Admitting that even a properly performed lumbar puncture may in exceptional cases hasten the course of a prognostically serious and often hopeless pathological condition found in intracranial hypertension (as in Case no. 425 of this series), this is not a just criterion by which to designate the procedure as unsafe ; rather, the accumulated experience of late years would justify the opinion that when properly done the procedure is reasonably safe, because of the low mortality—only two reported cases (Putnam [5]) in

recent years—and justifiably indicated because of the valuable diagnostic information it affords.

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XUM

MILITARY ASPECTS OF NARCOLEPSY, WITH REMARKS ON THE PATHOGENESIS OF NARCOLEPSY AND ON FATIGUE

By

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INTRODUCTION

An important by-product of Pavlov's work on cortical inhibition is the clarification of several hitherto puzzling problems of narcolepsy. I have already [3] considered the significance of Pavlov's work for such 'major' phenomena of narcolepsy as cataplexy and sleep-paralysis and for such 'minor' phenomena as the occurrence of sleep attacks in situations in which the patient must bear pain without flinching. In the present paper I shall endeavour to interpret certain clinical phenomena occurring in soldiers.

It is assumed that the reader is acquainted with Pavlov's views on internal inhibition and sleep. Briefly stated, Pavlov found that he could cause a conditioned reflex to undergo internal inhibition by using one of four methods. (1) *Experimental extinction:* This refers to the 'rapid and more or less smoothly progressive weakening of the reflex to a conditioned stimulus which is repeated a number of times without reinforcement' (Pavlov, p. 49). (2) *Conditioned inhibition:* This Pavlov defined as follows (p. 68): 'A positive conditioned stimulus is firmly established in a dog by means of the usual repetitions with reinforcement. A new stimulus is now occasionally added, and whenever the combination is applied, which may be at intervals sometimes extending to hours or days, it is never accompanied by the unconditioned stimulus. In this way the combination is gradually rendered ineffective, so that the conditioned stimulus when applied in combination with the additional stimulus loses its positive effect, although when applied singly and with constant reinforcement it retains its full powers.' (3) *Delay:* I omit giving details of this method. (4) *Differentiation:* E.g., a conditioned reflex is established to the tone of a tuning-fork of 800 double vibrations. The reflex having been established, it is now found that the tone of a fork of 812 d. v. will also evoke the reflex. If now the 812 stimulus is repeatedly applied *without* reinforcement, and the 800 repeatedly *with* reinforcement, the animal in the course of time differentiates the two stimuli, with the result that the reflex appears in response to the 800 but not to the 812. Pavlov showed the latter stimulus to have acquired inhibitory properties.

Of momentous significance is the fact that Pavlov found that experimental conditions which produced internal inhibition also (in many cases)

caused the animal to become drowsy and fall asleep (p. 251). He concluded that sleep is but widely irradiated internal inhibition.

FALLING ASLEEP ON SENTRY DUTY

The not infrequent occurrence of somnolence in sentries during the War aroused great curiosity among physicians. A common 'explanation' is that the sentry falls asleep because of the 'monotony' of his duties. This explanation, I shall try to show, puts the shoe on the wrong foot. One falls asleep, not 'because' of monotony, but because inhibition has irradiated over a sufficiently large part of the cortex. The environment, to be sure, is capable of checking this irradiation by bombarding one with excitatory (disinhibitory) stimuli. But to say that sleep is *caused* by insufficiency of excitation is as erroneous as to say that spasticity of the paralysed side in hemiplegia is caused by interruption of the pyramidal tract. Expressed properly, spasticity in hemiplegia represents the unopposed action of intact reflex arcs (Hughlings Jackson); similarly, falling asleep represents the unopposed spread of inhibition. We must therefore ask, does the work of a sentry favour the spread of inhibition? Consideration of the duties of a sentry will, I believe, show that this question must be answered in the affirmative.

Sentry duty constitutes a natural experiment which in all essential respects duplicates Pavlov's experiments on differentiation. The sentry has a conditioned defence reflex, describable thus: seeing (or otherwise perceiving) the enemy is followed by a response consisting either of aggressive movements or of movements serving to raise an alarm. The sentry must differentiate stimuli that really signify nearness of the enemy from those that represent only a 'false alarm.' He hears a faint rustling sound and 'is about to react' with a defence response; a moment later he realizes it was but the rustle of a nearby tree, whereupon a powerful inhibitory process checks the initial excitation. Let this happen a hundred times during the night and the cumulative effect of many inhibitory processes may well be such as to cause the soldier—especially if he have an unduly inhibitable cortex—to become irresistibly sleepy.

Using other words, we may say that sentry duty, necessitating an unusual degree of alertness, imposes an unusual strain on the cortex, thus encouraging cortical fatigue and its accompaniment, sleep.

MORBID REACTIONS DURING BOMBARDMENT

MacCurdy,[4] in his admirable article on War Neuroses, alluded to certain symptoms that are of the utmost importance for us here. I quote in full a paragraph beginning on the 16th page of his article (this paragraph describes the reactions of recruits to their first bombardment): 'The first actual trial which the recruit usually meets is the experience of being shelled. Naturally

the intensity of the bombardment varies greatly, and if the shells are falling at long intervals of time and at considerable distance, it is only the most unstable who are particularly affected. Few, if any, as far as one can learn, are absolutely normal on introduction to a heavy bombardment. By far the *commonest* response is one of fear, usually accompanied with the idea of running away, which the subject himself sees to be absurd. Although the men may make an effort to hide the signs of this fear, they are so frequently evident under initial shelling that the military authorities count on their appearance. Their presence at this time is no indication of the degree of indifference which may later develop. A *less common* reaction is that of *excitement*, accompanied even with a kind of *spurious elation*. The man has a tendency to make facetious remarks about the shells, to laugh at feeble witticisms, and very often feels under considerable motor tension, there being a pressing desire to do something, to do it immediately and do it hard. A *still more unusual* but very interesting reaction is that of *slowness or languor* (which may succeed primary fear). This may be accompanied by a depressive affect or by *lethargy so extreme that the individual will lie down and go perforce to sleep* with a pathological indifference to the danger ' (italics mine).

Let us consider these phenomena from the viewpoint of Pavlov's work. When shells are falling about us, the ' natural tendency ' is to run away—a tendency which is suppressed because of the desire not to appear a coward. This situation offers us a perfect instance of conditioned inhibition : an action which is permissible when the stimulus consists just of flying missiles (e.g., when dynamite explodes near us in civil life) becomes forbidden when, so to speak, the stimulus consists of flying missiles *plus* the environment of a battlefield. When we consider the strength of the defence reflex to pain, we may plausibly suppose that it is a powerful inhibitory process that keeps soldiers from running away at their first bombardment. Herein, I believe, lies the explanation of the somnolence noted by MacCurdy in some soldiers.

MacCurdy found that some soldiers, instead of being sleepy, reacted with ' a kind of spurious elation,' with a tendency to ' laugh at feeble witticisms,' etc. These phenomena are, I submit, due to inhibition of the highest layers of the highest centres (Hughlings Jackson), and bear the same relation to somnolence that the elation of the mildly intoxicated man bears to the stupor of the profoundly intoxicated man. In discussing the problem of varying depths of dissolution, Jackson argued that a small dose of alcohol paralyses only the highest layer of the highest centres, leaving the subject comparatively competent but lacking self-control, wherefore he is elated and extravagant ; a very large dose paralyses *all* of the highest centres and some of the lower centres as well, leaving the subject very incompetent. We may justly argue by analogy that in the spuriously elated soldier there is inhibition limited to the topmost layers of the highest centres, while in him who falls asleep during bombardment there is much more widespread inhibition. This statement accords with MacCurdy's observation that spurious elation is a

commoner reaction than somnolence: we may rightly expect the milder form of a morbid phenomenon to occur more commonly than the advanced form.

I digress to call attention to several cases of narcolepsy in which, in civil life, the patient fell asleep in situations in which he must have been conscious of danger. Hilpert's patient fell asleep while working on a scaffold, as did also the patient in Spiller's Case I. In Spiller's Case II the patient, a locomotive fireman, fell asleep ' while holding on to the grabirons of a train said to be moving at a speed of about 70 miles an hour.' In Wilson's Case I the patient, a railway carpenter, had a sleep attack ' when actually on the top of a signal post executing a repair.' In these cases we may say that the ' natural tendency ' to escape from a position of danger was inhibited, as a result of which the (easily inhibitable) brain of the patient underwent widespread inhibition. The mechanism provocative of sleep attacks in the four patients cited in this paragraph seems identical with that operative in the soldiers who fell asleep during their first bombardment.

In my former paper [3] I considered two cases in which the patient fell asleep while suffering pain. One was a narcoleptic patient of mine who fell asleep while the dentist was drilling his teeth; the other was a patient of Gowers' who fell asleep while a probe was being passed down a nasal fistula. I pointed out that the significant feature of these cases was that the patients, as self-respecting adults, were obliged to bear the pain without flinching, i.e., they had to suppress their defence reflex to pain. I also alluded to one of Rothfeld's cases, in which the voluntary suppression of sexual reflexes precipitated an attack of a certain form of ' localized sleep.' We may make the general statement that *in persons with easily inhibitable brains the voluntary suppression of powerful reflexes may cause the appearance of localized or generalized sleep.* All the cases mentioned in this section represent special instances of this general phenomenon.

FATIGUE

MacCurdy called attention to the fact that war neuroses were unknown prior to the era of modern trench warfare, and concluded that the latter is a necessary cause of the former. In analysing the nature of modern warfare, he said (p. 12): ' Anyone who is at all familiar with the phenomena of modern trench warfare can see that it makes a great demand on the devotion of the belligerents and offers little personal satisfaction in return. In previous wars the soldiers, it is true, were called upon to suffer fatigue and expose themselves to great danger. In return, however, they were compensated by the excitement of more active operations, the more frequent possibility of gaining some satisfaction in active hand-to-hand fighting, where they might feel the joy of personal prowess. Now, the soldier must remain for days, weeks, even months, in a narrow trench or a stuffy dugout, exposed to a constant danger of the most fearful kind, namely, bombardment with high explosive shells which

come from some unseen source, and against which no personal agility or wit is of any avail. This naturally occasions great fatigue, and, on the other hand, opportunities of active hand-to-hand fighting are rare, so that a man may be exposed for months to the appalling effects of bombardment and never once have a chance to retaliate in a personal way. Consequently the sublimations described above are more difficult to maintain than in any previous war. The soldier becomes fatigued . . .'

MacCurdy wrote this 10 years before Pavlov's work became generally known in western Europe, and it is remarkable that he surmised, between fatigue and the suppression of the instinct for hand-to-hand fighting, a relationship which Pavlov's work plausibly explains. Being at the front arouses in the soldier the ' primitive expectation ' of physically meeting an antagonist, exactly as in an appropriately trained dog the sound of a metronome arouses the expectation of a dish of meat. When in the dog the conditioned stimulus is no longer reinforced (i.e., when the sound of the metronome is no longer followed by the presentation of meat), an inhibitory process occurs and the conditioned reflex undergoes ' experimental extinction ' (i.e., the sound of the metronome no longer excites the flow of saliva). These conditions are duplicated in the case of the soldier, who, in spite of his ' primitive expectation,' has no physical meeting with an antagonist. In consequence, just as in the dog the flow of saliva is inhibited, so in the soldier the movements of hand-to-hand fighting are inhibited. Since dogs, during experimental extinction, frequently get drowsy and fall asleep, we may attach significance to the occurrence of fatigue in soldiers under like conditions ; *fatigue under these conditions is a manifestation of inhibition.*

Both military and civil life offers countless situations in which one must suppress strong impulses. The impulse to run away during bombardment has already been alluded to. MacCurdy was impressed by the apparent importance, as a fatigue-provoking factor, of the situation of the soldier or officer who finds himself under an antagonistic or incompetent superior whose unfair or unwise orders he may not question (see, e.g., his Cases X and XI). In this situation the soldier must suppress his impulse to protest, exactly as during bombardment he must suppress his impulse to run away. As a civil counterpart, I cite the instance of a clergyman of 65, a man of unusual physical and mental vigour. (He consulted me for no infirmity and I know him only as a friend.) A man of great integrity, he finds among his parishioners much hypocrisy, which he would vigorously denounce if he were financially independent. He has repeatedly observed that after a sermon touching on topics which more than ordinarily stimulate his denunciatory impulses, thus necessitating on his part an unusual effort at suppression, he is fatigued to the point of exhaustion, while on the other hand a brisk five-mile walk, which he takes daily, causes, in spite of his age, almost no fatigue.

In the cases cited in the last paragraph, we deal with conditioned

inhibition. The mechanism of these cases differs from that of the soldiers who lack an outlet for their impulse for hand-to-hand fighting.

MacCurdy observed that ' the first sign of an approaching neurosis is fatigue ' (p. 18). We may therefore regard fatigue as a relatively *mild* clinical manifestation of inhibition.

I beg the reader to note that I make no general statement about all cases of fatigue, but assert only that in *some* cases it seems proper to regard fatigue as a manifestation of cortical inhibition produced by mechanisms identical with those revealed by Pavlov's experiments.

HYPNAGOGIC HALLUCINATIONS

MacCurdy found that the early stages of war neuroses were characterized, in addition to diurnal fatigue, by hypnagogic hallucinations. He wrote (p. 20) : ' There is great difficulty in getting to sleep, with a long period of hypnagogic hallucinations. Whatever has been the dominating experience of the day appears in troublesome vision before the eyes of the soldier, who, although knowing that what he sees is not actually there, is still unable either to go to sleep or to awaken himself sufficiently to banish the visions.'

To discuss adequately the physical processes concomitant with hallucinations would carry us beyond the scope of this paper. It will suffice to say that there is good reason to believe that hypnagogic hallucinations indicate a *partial* inhibition of the cortex. I have already called attention [3] to the remarkable frequency with which hallucinations occur during sleep-paralysis, a state in which the patient, on falling asleep or awaking from sleep, is conscious but paralysed. In sleep-paralysis *without* hallucinations, inhibition is localized in the motility substrate, the ' substrate of consciousness ' being (so far as we can now determine) free of inhibition ; in sleep-paralysis *with* hallucinations there is, besides the complete inhibition of the motility substrate, *partial* inhibition of the substrate of consciousness (not *complete* inhibition of the substrate of consciousness, because in that case the patient would be fully unconscious, i.e., asleep). Hypnagogic hallucinations in the soldier at the front are, therefore, cognate to diurnal fatigue, in that both phenomena are manifestations of inhibition.

The reader may ask, Since it is claimed that trench warfare encourages cortical inhibition, and since inhibition and sleep are manifestations of the same thing, why does not the neurotic soldier sleep soundly at night instead of poorly ? The answer to this question is for the present unknown, as is also that to the question of why narcoleptics in civil life so often sleep poorly at night, contrary to the expectation that because they suffer from somnolence they will therefore sleep unusually soundly.

INHIBITORY PHENOMENA BEHIND THE BATTLE-FRONT

While the battle-front offers the easiest opportunities for the production of inhibition, military service behind the front is not without such opportuni-

ties. Collins' Case I, in which the patient's narcolepsy began while he was in the army, shows remarkably well the apparent relationship that may exist between military service and narcolepsy. Because of its great interest I cite Collins' description of the origin of his patient's symptoms: 'He noticed that while drilling he would become very tired. As time went on he would fall over while drilling because of extreme weakness in his muscles. When he went in for mess at noon he would fall asleep and it was with the greatest difficulty that the sergeant could awaken him for afternoon duty.'

It is obvious that on entering the army the recruit must radically alter the whole mode of his life, i.e., he must discard (inhibit) many of the conditioned reflexes of civil life and acquire many new ones. Thus the responses of the recruit to the commands of the drill-sergeant represent a set of conditioned reflexes. Now, in addition to the four methods whereby Pavlov found he could artificially induce inhibition, there is a spontaneous inhibition which conditioned reflexes undergo *even when they are regularly reinforced*. According to Pavlov (p. 234), 'one of the most important' properties of cortical cells is that 'under the influence of conditioned stimuli they pass, sooner or later, into inhibition.' After citing a number of experiments, he stated (p. 244): 'The fundamental fact in all these experiments . . . is the transition sooner or later into inhibition of the state of the cortical elements acted upon by the conditioned stimulus. So far as concerns all the experimental evidence at our disposal up to the present, this transition must be regarded as depending on a functional exhaustion of the cortical elements as a result of their activity in response to a stimulus.' It is therefore understandable that long daily drill-periods continued for many months should encourage the occurrence of inhibitory phenomena, and the onset of narcolepsy during a period of military training in Collins' case should not be hastily dismissed as a coincidence.

Should there be another war, it would be desirable to study carefully the relation of narcolepsy to military service. It would be desirable to ascertain how many cases begin in training, and how soon after entrance into the army they begin. It would be desirable to make a careful study of fatigue, morbid elation, hypnagogic hallucinations, nocturnal restlessness, cataplexy, sleep-paralysis and morbid somnolence, with especial attention to the types of situation that seem to encourage the appearance of these phenomena.

SUMMARY

Certain situations in military life constitute natural experiments in which are duplicated the essential conditions of Pavlov's laboratory experiments on internal inhibition. Some of these situations are: (1) trench warfare, in which the impulse for hand-to-hand fighting is inhibited; (2) bombardment, in which the impulse to run away is inhibited; (3) sentry duty, in which the sentry must differentiate insignificant stimuli from those that indicate the

approach of the enemy. The occurrence of narcoleptic symptoms in response to these situations is a phenomenon identical with the occurrence of inhibition and sleep in Pavlov's corresponding experiments. Consideration is given to the occurrence, in these situations, of morbid somnolence, hypnagogic hallucinations, fatigue and morbid elation.

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SOME RELATIONSHIPS BETWEEN PERSONALITY AND BODY CHEMISTRY *

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INTRODUCTION

With the tremendous advance of biochemistry in recent years, physiology has found it advantageous to direct both its methods of research and its explanatory concepts along chemical lines. Physiological psychology has been somewhat slower to adopt this new point of view. But a perusal of the literature of the past decade soon shows that it is gradually falling into line. There has been a slowly increasing number of studies relating various aspects of behaviour to chemical changes within the body. Research in this field has been following, in the main, four principal directions: (1) endocrine research, which includes extirpation and feeding experiments as well as the data derived from the endocrinopathies; (2) the effects of various types of activity, intellectual, volitional or emotional, upon metabolism, secretion and body constitution; (3) the effects of drugs and chemicals upon behaviour; and (4) studies of the correlation between the chemistry of the body, including its excretions, and those patterns of total behaviour which we call personality.

It is with this fourth type of psychobiochemical study that this paper is concerned. Although its beginnings can readily be traced back a quarter of a century (and even older investigations pointing out the direction of future endeavour are to be found), the real development of the field has been within our own time. Like so many other branches of human biology, the first impetus seems to have come from those who work with disease. For a number of years there has been considerable interest in the differential chemistry of those gross deviations of personality, the psychoses and neuroses. More recently attempts have been made to extend these investigations to the study of personality variations which are within the range of normality.

ACID-BASE BALANCE

One of the most fruitful fields of research has been the acid-base balance of the body. Ludlum was among the first to arouse interest in its possibilities. After he and his co-workers had found that there was a differentiation between excited and confused types in acute insanity on the basis of the reaction of the secretions and excretions of the body, they proceeded, perhaps somewhat

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prematurely, to relate gross neuromuscular symptom-pictures to the two types.[1,2,3,4] The theoretical implications are both suggestive and stimulating, even though factual data are absent.

Another series of observations bearing upon the problem has come from the Psychological Clinic at Philadelphia. Starr,[5] who was the pioneer in this work, was able to show a marked difference in the hydrogen-ion content of saliva as between the so-called ' psychopathic ' and ' sub-breather ' types of stammerer, with a change in the saliva of the latter as he was freed under treatment from the acidosis due to the defective respiratory elimination of carbon dioxide. Later, it was found simpler to study the carbon-dioxide content of alveolar air, and similar results were obtained.[6] In clinic patients other than stammerers, similar relationships have been found.[7] Children who were apathetic were found to be mildly acidotic, due to inefficient respiratory elimination.

This point of view has been further emphasized by a recent group of studies from the laboratory of the London County Asylums.[8,9,10,11] The workers there find evidence of a rather definite disturbance in the acid-base equilibrium in psychotics. The normal tides in urinary excretion following the ingestion of acid or alkali are absent in these patients, who tend to maintain a flat level of urinary acidity, or to reverse the normal tides. In melancholics the urine tends to a constant acidity, with low alkali reserve of the blood. They have come to the conclusion that these abnormalities are due to inadequacy of the respiratory, rather than of the urinary, mechanism for acid-base regulation. In support of this contention it is shown that when 2 per cent. of carbon dioxide is breathed the increase in respiratory activity so noticeable in the normal subject is absent in the psychotic.[12] Moreover, the administration of sodium bicarbonate causes increased urinary alkalinity in both types of subject, but the psychotic fails to show the normal curve for the rise and fall of the carbon dioxide content of alveolar air. These results tend to be somewhat confusing, however, because various types of psychoses are thrown together to form a non-homogeneous group.

In our own studies, we have worked with individuals who were, in general, within the range of normality. Here the differentiation of personality patterns has depended not upon the gross distinctions of mental disease, but upon the finer, and therefore less accurately determinable, gradations measured by a rating scale. We now have at hand ample data to show that, even with the crude procedures employed, there is a definite positive correlation between emotional excitability and relative alkalinity of the saliva.[13,14] The more excitable individual tends to have a less acid saliva than does the calm and apathetic person. The same tendency is also evident in determinations of urinary acidity. The apathetic individual excretes by this channel more acid than does the emotionally excitable person.

So far the data all point in the same direction. The evidence may be summed up as showing that in the gross personality-deviations of mental

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disease there is some disturbance of the acid-base balance, and that, by and large, lowered acidity tends to be associated with the more active states and heightened acidity with the more apathetic conditions.

But a seeming exception is at once apparent. The effects of carbon dioxide inhalation upon catatonic patients are well-known. We may at once ask how it is that, if carbon dioxide retention is associated with body acidity and emotional dullness, the inhalation of large amounts of this gas causes the catatonic or postencephalitic patient to become more active. If, however, one examines further, it is apparent that during the experimental inhalation there are several other factors at work. One is the presence of oxygen to the extent of a very high partial pressure with, as Freeman [15] points out, increased oxidative processes in the brain. Another is the rapid respiratory rate, which creates a situation quite different from that which exists when an individual is respiratorily sluggish and therefore retains carbon dioxide. Moreover, similar effects with respect to activation of the patient can be produced by certain narcotic agents, which make the patient even more sluggish than does carbon dioxide. Our present knowledge of the mechanism acting in bringing about these results is too vague to permit us to harmonize them with the other observations which we have quoted.

We now come to the question of possible interpretation and explanation of the data already at hand with respect to the rôle of the acid-base equilibrium in connection with personality. The most widely accepted view makes respiratory sluggishness the prime factor in the picture. According to this view, the person who fails to breathe properly because of lowered activity (or perhaps a lowered threshold) of the respiratory centre is unable to eliminate carbon dioxide properly. His alveolar air shows a high percentage of this gas. His urine is acid, because the urinary mechanism must help to maintain the balance. His saliva tends toward acidity. In addition, he may be emotionally flat, apathetic or even lethargic. We must postulate, therefore, that retained carbon dioxide acts as a drug, a depressant of cortical and thalamic activity.

Another theory, that of Armstrong,[16] is that the carbon dioxide in the blood plasma regulates the flow of chlorides from the plasma to the corpuscles and vice versâ. When a normal person awakes and increases his respiratory ventilation there is increased excretion of chlorides in the urine. But in psychotics there is retention of chlorine, and in catatonics an actual reversal of the chloride shift, because their pulmonary ventilation is greater in sleep than while awake. Further data are desirable before we can evaluate these findings. More detailed study of chloride metabolism is indicated as a direction for future research in this field.

CREATININE

In connexion with the above considerations, we may next turn our attention to the relations of creatinine metabolism to personality charac-

teristics. It is commonly accepted in biochemistry that creatinine is a product of muscular metabolism and that the amount of it formed and excreted depends upon the size and development, but not the activity, of the muscular system. This point of view naturally turned the attention of psychiatrists to the study of creatinine in connection with patients who show disorders in activity. But when Weinberg [17] investigated the creatinine output of patients with neuromuscular diseases he was led to the conclusion that such variations as he found were due not to the disturbances in muscular tonus, but to mental influences. Soon afterwards Looney [18] found marked increases in the creatinine content of the blood of catatonic patients, while Stratton [19] reported a detailed study of the changes in the creatinine excretion of a sub-breathing stammerer under treatment and in relapses.

Working, as before, with personality differences which are within the range of normality, we have found a relationship of emotional excitability to creatinine production somewhat parallelling that to body acidity. Our correlations [13] show that the most excitable persons tend to have both the lowest concentration of creatinine in the blood and the smallest relative amounts of excretion of creatinine in the urine. These results fit in with the observations on the stammerer and also with the findings in catatonics, since the latter condition may be considered as one of decreased liability to emotional response.

It is rather interesting that both creatinine excretion and urinary acidity tend to vary with the person's state of emotional lability. But it is not so surprising when one notes, as we [20] have shown elsewhere, that these two urinary measurements have a high tendency to vary concomitantly. This relationship opens several possibilities of explanation. Are the variations in creatinine output entirely secondary to those in the acid-base equilibrium? Does the state of the central nervous system profoundly affect muscular metabolism? Should we speak of personality, considered as gross patterns of behaviour, as dependent upon the condition of the neuromuscular system rather than that of the nervous system? Or must we consider the relationship as one due to the action of creatinine as a drug which modifies behaviour? The answers to these questions are still in the future.

PHOSPHORUS

The relation of phosphorus to mental activity has long intrigued the popular mind. Yet it appears to have been of little interest to the scientists. There have been a few observations on the phosphorus excretion of psychotics, with almost uniformly negative results. A striking exception is the work of Folin and Schaefer,[21] who studied a single case of manic-depressive insanity and found a consistent alternation of the nitrogen-phosphate and sulphate-phosphate ratios in the urine as between normal and disturbed periods. They attributed the difference to an abnormality in the assimilation of the phosphorus. More recently Powers [22] has claimed to find an increase above

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the normal in the phosphorus content of the blood of idiots, but another experimenter, Negus,[23] has failed to find this difference.

Our own experiments [13] have produced markedly discordant results with respect to phosphorus. Thus the personality characteristic of good-naturedness correlates positively with the urinary excretion of phosphorus and negatively with the amount of phosphorus in the blood. Nor did a more detailed study [24] of the various types of phosphorus in the blood clarify the situation, but, on the contrary, added to the confusion. Even the most marked correspondence, i.e., the highest correlation, found in the entire experiment, which was between intelligence and phosphorus excretion, was determined in so small a group as to be of doubtful significance. Although it is in accord with the findings of Powers in the case of the blood phosphorus of idiots, as mentioned above, our figures for the phosphorus of the blood agree with neither. These fragmentary and contradictory results make it impossible to come to any conclusion as to the relation of phosphorus to personality traits, but they do suggest further study of the subject.

CALCIUM

A number of recent investigations of the effect of various ions upon living tissue, and particularly upon the peripheral nerves, has aroused interest in the rôle of calcium. Yet it is surprising to find that as yet no relationship between personality and the calcium content of body fluids has come to light. The few studies of persons with mental disease yield nothing that is consistent. The experiments [24] upon normal individuals have not shown correlations between the calcium of the blood and such personality traits as good-naturedness, aggressiveness, emotional excitability or intelligence of sufficient size to be considered reliable. Here, again, further study is indicated.

CONCLUSIONS

No attempt has been made in this paper to make a comprehensive survey of all the studies which relate personality, normal or abnormal, to the chemical activity of the body. Thus the growing literature which links up blood-sugar curves, basal metabolism, nitrogenous constituents and products of the body, and, quite recently, colloid chemistry to behaviour has not been touched upon. We have considered in some detail four typical biochemical conditions and their relations to personality. In the case of acid-base equilibrium and creatinine production, it would seem that at the present time sufficient observations, obtained by various methods and from various points of view, are now at hand to enable us definitely to relate both of these chemical factors to the individual's emotional excitability. Much, of course, remains to be done. As has been already noted, in neither case is the explanatory interpretation clear. The choice between various hypotheses must await further facts. Nor are all the facts as yet unequivocal. In the case of phosphorus, the results which have been obtained as yet are so discordant

as to lead to no rational interpretation of any consistent relationship. Yet they are sufficiently suggestive to incite one to further investigation. Finally, we noted that calcium, which a priori would have been expected to yield significant results, has led only to negative findings.

With these facts in mind, one may well ask whether or not investigations of the sort which we have quoted are worth while. At first sight it seems that the positive results are meagre. But it must be remembered that research in this field is quite new, and that few workers have been engaged in it. Moreover, the field is tremendous in extent and only a few of the many possibilities have even been touched upon. If these considerations are kept in mind, the situation seems somewhat different. The findings are not really so few in proportion to the amount of work done. They compare, from this point of view, not so unfavourably with those obtained by workers who have followed the other lines of psychobiochemical investigation mentioned at the beginning of the paper, not to mention the non-chemical field of endeavour in physiological psychology. It is, perhaps, permissible to conclude with a feeling of optimism as to the future of research into the relations of personality to body chemistry.

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Editorial

HEREDITY IN PSYCHIATRY

THE recent controversy started on eugenic grounds regarding the inheritance of mental deficiency has served at least one useful purpose. It has drawn attention to the almost complete absence of data of a scientifically acceptable kind from any source in this country on the heredity of mental defect. Such statistics as were available in the textbooks and journals covered an insignificant total of cases, and have not been collected in a way that would bring satisfaction to a geneticist. The same is true of work abroad in this particular field but not to the same extent. It does not very well become us to criticize some of the foreign work because it seems extravagant. Only in the past three years or so has any sound attempt been made in this country to fill the gap with regard to the inheritance of mental deficiency on well-considered genetic principles.

With mental disease as with mental deficiency, the position in this country is also unsatisfactory. In the technical journals, which presumably reflect in their contents the leading interests of those engaged in research, it is a rare event to find a paper on the inheritance of mental disease. Yet the opportunities are very great. Clouston, many years ago, set an example of what might be done. '. . . An inquiry I made into the history of 83 families, taken at random, in a country parish in Scotland, all the members of which were personally known to me for three generations. I took every family I really knew and no others. I made no selection. They were all country people, decent folks, hard-working, thrifty, few very poor, indeed mostly money-loving, with scarcely any drunkards.

'I took advantage of my intimate knowledge of the people as a doctor to count in the lesser attacks of melancholia, the milder delusional states and the milder degrees of imbecility, that would not have gone into any public statistics of lunacy or census schedules. Still, they were all mental diseases or defects just as truly, from the scientific point of view, as our acutest case in Morningside. The result was this, that of those 83

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families, I knew that in 41 of them one or other of those four diseases had occurred.'

The great majority of our mental hospitals draw on a local non-migratory population resembling that available to Clouston on a much larger scale. A high proportion of the families on which they draw have been settled in the same district for several generations, presenting opportunities that can seldom be surpassed ; yet for our more thorough information at present we have to go abroad. While we are surprised and disturbed by some of the conclusions reached by our foreign colleagues, we have little of our own except clinical impressionism to fall back upon.

This lack of interest in heredity, judging from the published material, justifies pointing out what should be obvious, that hereditary data are most valuable and sometimes essential in daily medical practice. Medical students do not have it emphasized enough to them how important the information about a patient's heredity can be for diagnosis, especially in difficult cases. The old type of doctor was credited with a knowledge of the constitution of his patient. How much of this must have been compounded of a knowledge of the disease-tendencies of their families whom they looked after often through two or even three generations ? Mental constitution, apart from mental disease, is a more or less important factor in almost any morbid condition. It has familial and hereditary aspects which are more readily grasped through the study of those exaggerated traits which are transmitted through successive generations, appearing in various combinations in individual members, of families tainted with mental disease. Studies of the inheritance of characterological and temperamental traits of this type, that is, the type usually credited with close association with or predisposition to actual mental disorder, is one of the most interesting by-products of the recent work on constitution with which Kretschmer's name is so much associated. It throws light conversely on what constitutes a unitary trait of character and temperament and thereby illumines a very difficult field of psychopathology.

What has been partly done for the psychoses in this way badly needs extending to the psychoneuroses. Even those psychopathologists who claim to reduce any psychoneurosis to ultimate immediately postnatal terms are forced to postulate a constitutional factor for anxiety, to take an example. A study of the inheritance of such a supposed constitutional factor can show how much it matters. There can be little

doubt that psychopathologists in general sometimes set themselves too hard a task which a better knowledge of the constitutional element and of the weight to be assigned to it would teach them to undertake not so optimistically. Thereby they offer themselves too willing victims to that excessive demand made by the public of medicine in general, namely, to make bricks without straw and to give mental or physical 'stuff' and a resistance to disease that the patient has never been given the opportunity by his ancestors to possess. And thus psychological medicine, especially, sometimes comes into unnecessary disrepute.

There are certain offshoots of any investigation into the inheritance of mental disease which are of interest and might prove fruitful topics for research. The hereditary aspects of allergy have always been appreciated, as, for example, with the eczema-asthma-prurigo complex, but there is an analogous field for investigation in hereditary idiosyncrasies to certain drugs. That individual idiosyncrasy exists with regard to the barbiturates is well known, and that they can have something like a specific action on anxiety is also well known. It would be very interesting to know whether such drug idiosyncrasies are heritable. It seems possible from fragmentary clinical observation that something of interest might emerge; for example, it has even been occasionally observed that a family hypersensitivity to bromide exists. Knowledge of the hereditary aspects of nervous temperaments and constitutions might be increased in this way.

The true neuroses—that is, functional disturbances which are not apparently psychogenic—form another domain which overlaps that of the hereditary psychopathies. It seems that the vegetative functions, in spite of Langley's brilliant pioneer work, have received less attention clinically here than abroad, and probably much less attention than they deserve. Everyone knows how obscure disturbances seemingly subjective, but not easily explained on purely psychological grounds, are sometimes found, for instance, in association with an epileptic 'anlage.' It seems that in many of these obscure disturbances where there is no physical disease which is relevant, a study of the hereditary relationships, as Alvarez has recently pointed out, especially in the mental field, often gives a clue to what would otherwise remain a mysterious disorder, or might readily be mistaken for something else and given erroneous and unfortunate treatment. Work undertaken along these or similar lines would not only add to our fund of knowledge, but would

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tend gradually to produce an influence on the teaching of medicine, so that the present trend of seeing the patient as a whole, which is gaining momentum, would be reinforced by a sane philosophical viewpoint of the patient as a whole against the background of his ancestry.

Abstracts

Neurology

NEURO-ANATOMY AND NEUROPHYSIOLOGY

[43] **The development and myelination of the posterior longitudinal bundle in the human.**—M. F. Lucas Keene and E. E. Hewer. *Jour. of Anat.*, 1933, **77**, 522.

The principal results of this anatomical research can be summarized as follows :

The bundle is one of the earliest tracts to myelinate, beginning at about 14 weeks. It first appears as two unconnected groups of fibres, one set related to Deiters' nucleus and extending upwards to the lower pons and downward to the medulla and cord ; the other is found in the upper pons passing to the fourth nerve nucleus. As growth proceeds in the fœtus myelinated connexions develop between the portions of the bundle. Four nuclei can be identified in connexion with it. The nucleus of the posterior commissure makes a definite connexion with the bundle and furnishes a large contingent of fibres to the ventral part of the commissure. The interstitial nucleus of Cajal, and the nucleus of Darkschewitsch, form part of the nuclear system.

J. V.

[44] **The arterial irrigation of the cerebellar nuclei in man** (L'irrorazione arteriosa dei nuclei cerebellari nell' uomo).—I. Fazzari. *Riv. di pat. nerv. e ment.*, 1933, **41**, 558.

An investigation, by dissection and microscopical inspection of vessels injected with coloured material, of the origin and distribution of arteries to the cerebellar nuclei. Both anterior and posterior branches go to the nuclei from the anterior cerebellar artery and the vertebro-cerebellar arteries respectively. The field of distribution seems to correspond with the ontogenetic and phylogenetic development of the various parts of the nuclei. The type of the distribution of the vessels is different in the different zones of the nuclei.

R. G. G.

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[45] **Old and new studies on the centrifugal fibres of the posterior roots** (Anciennes et nouvelles recherches sur les fibres centrifuges des racines postérieures).—E. LUGARO. *Arch. Suisses Neurol. et Psychiat.*, 1933, **31,** 284.

IN a beautifully illustrated article Professor Lugaro shows that in the posterior roots, within a period of some 60 to 100 days after extirpation of several of the posterior root ganglia, fine myelinated fibres can be seen. Apart from occasional aberrant fibres, his conclusion is that these centrifugal fibres have regenerated, and come from neighbouring tissues. This work of his was done in 1906 and apparently ignored. But recently similar experiments have been performed by others, and the conclusions are conflicting. Professor Lugaro now states, after fresh experimentation, that the genuinely centrifugal fibres of the posterior roots, emerging from the cord and ' born ' in it, are amyelinic, and that there are no centrifugal myelinated fibres in the roots. Those which, as he originally discovered, are preserved after removal of root ganglia belong to adjoining roots which have not been involved, and have been ' borrowed ' from them ; others which, as remarked already, are found later, are not pre-existing normal fibres, but have regenerated and penetrated the cicatrix of the root concerned. He allows the occurrence of numerous centripetal amyelinic fibres issuing from ' obscure ' cells in the root ganglia.

S. A. K. W.

[46] **The development of Hortega's microglia** (Weitere Untersuchungen über die Entwicklung der Hortegaschen Mikroglia).—H. v. SANTHA and A. JUBA. *Archiv f. Psychiat.*, 1932, **98,** 598.

THE conclusions of this finely illustrated article, embodying the results of researches on rat fœtuses, may be summarized as follows :—

There can now scarcely be any doubt that microglia must developmentally be sharply distinguished from neuroglia. Nor can it be doubted that its genesis is closely linked to vascularization of the central nervous system. The very first appearance of microglia is associated with the first traces of vascular growth, and before that stage is reached the neural parenchyma is free of microglia. The retina is apparently the sole neural tissue where microglial cells develop in the absence of vessels, yet in all probability the cells immigrate from the capillaries of the vitreous. Further, evidence is offered which goes to show that cells indistinguishable from those of the microglia of the nervous system are to be found in the heart, bladder and other organs.

S. A. K. W.

[47] **The place of the red nucleus in the postural complex.**—W. R. INGRAM and S. W. RANSON. *Amer. Jour. Physiol.*, 1932, **102**, 466.

COMPLETE destruction of the red nuclei or their descending connexions in the cat by restricted lesions leaves the animal capable of righting itself, standing and walking, even after optic control has been removed (by enucleation or blindfolding). Hence the red nucleus cannot be regarded as an entirely essential centre for the righting function in blind animals. Dysmetria and ataxia or incoordination are often found in such animals with red-nucleus lesions, perhaps due to involvement of the cerebellar efferent system. Mild extensor hypertonus is also present.

The red nucleus cannot alone be responsible for inhibitory influences, removal of which causes decerebrate rigidity; it is merely a portion of the complex system regulating postural functions.

A. B.

[48] **Role of the anterior roots in visceral sensibility.**—THEODORE T. STONE. *Arch. of Neurol. and Psychiat.*, 1933, **30**, 99.

IN the past twenty-five years the anterior roots have been said by many investigators to contain antidromic fibres which conduct painful impulses from the viscera and skin. The purpose of this experimental study was to determine whether or not there are antidromic fibres in the anterior roots which serve to transmit sensations.

The evidence from the experimental work detailed shows that there are no antidromic fibres for pain in the anterior roots in relation to visceral sensibility. Investigators who have previously stated that there must be antidromic sensory fibres in the anterior roots may have been led to such a conclusion because of the following possibilities: first, when the posterior roots were intact, stimulation of the anterior roots produced pain because of the nervi nervorum to these roots. Second, when pain persisted after section of the posterior roots, an insufficient number of roots may have been severed. Third, movements due to fright and other causes may have been interpreted as pain. Fourth, when the anterior roots alone were severed, hæmorrhage and trauma may have resulted in injury to the posterior roots, or changes may have occurred in the spinal cord at some distance. Fifth, at autopsy one may not find as many roots severed as were purported to be.

It would seem that in such visceral painful impulses as are produced by distension of the gallbladder the anterior roots are in no way a conduction pathway. It is believed, therefore, that the pathway for visceral afferent impulses from the gallbladder passes upward by the right splanchnic nerve through the thoracic sympathetic trunk to the spinal nerves by way of the white rami and then into the spinal cord by way of the posterior roots.

R. M. S.

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NEUROPATHOLOGY

[49] **An improved colorimetric method for the determination of the bromide content of body fluids.**—HENRY TOD. *Jour. of Ment. Sci.*, 1933, **79**, 373.

AN improved colorimetric method is described for the determination of the bromide content of body fluids, requiring 2 c.c. of the fluid. It is suggested that this method may be of use in ascertaining rapidly whether a confused or delirious state is due to bromide intoxication or not.

C. S. R.

[50] **Müller's reaction in the cerebrospinal fluid** (La reazione di Müller nel liquido cerebro-spinale).—N. SACCHETTI. *Riv. di pat. nerv. e ment.*, 1932, **40**, 94.

MÜLLER'S reaction is a flocculation reaction, and this was compared with the Wassermann reaction in 194 patients suffering partly from neurosyphilis or late syphilis, partly from non-syphilitic nervous or mental diseases, and partly healthy. The author claims a greater sensitivity for Müller's reaction, getting positives in certain meningitic cases which gave negative Wassermann reactions, and finding that cases treated with malaria retained a positive Müller's reaction longer than the Wassermann reaction. On the other hand, he does not consider Müller's reaction or other flocculation reactions have the value enjoyed by the Wassermann reaction, as modified by Hauptmann, in differentiating general paralysis from other neurosyphilitic conditions.

R. G. G.

[51] **The quotient of meningeal permeability to normal buffer hæmolytic amboceptor in cerebrospinal syphilis** (Quoziente di permeabilita meningea al normale ambocettore emolitico antimontone nella lue del sistema nervoso).—M. PIOLTI. *Riv. di pat. nerv. e ment.*, 1932, **40**, 390.

THE method is explained and the findings of 26 cases of progressive paresis are recorded. The result was positive and variable between 0·2 and 1 in 100 per cent. of cases; in 10 per cent. of these the presence of complement was also demonstrated. The sensibility of this test in paresis is superior to that of the Wassermann test, and in the group of specific affections of the nervous system is pathognomonic of paresis and tabes. Malarial therapy lowers and sometimes abolishes the quotient, but clinical improvement does not always correspond to the improvement in the quotient. This quotient is the only one which differentiates between paresis and cerebral syphilis. It

is therefore specially useful and may help to further the problem of the biological differentiation between these two.

R. G. G.

[52] **The spinal fluid and its pressure in cerebral hæmorrhage and thrombosis** (Recherches sur la pression rachidienne et sur le liquide céphalo-rachidien dans les hémorrhagies et les thromboses du cerveau).—K. H. Krabbe and E. Geert-Jorgensen. *Acta Psychiat. et Neurol.*, 1931, **6**, 529.

Some 140 cases of cerebral hæmorrhage or thrombosis (with 50 autopsies) were studied completely as regards spinal fluid pressure and contents.

A spinal pressure above 300 is suggestive of hæmorrhage, but does not exclude thrombosis.

Hæmatorrhachis or xanthochromia of the fluid points with all but complete certainty to cerebral or meningeal hæmorrhage, except in those rare cases where thrombosis has induced a hæmorrhagic infarct.

Increase of protein above 20 indicates hæmorrhage, though the fluid may not be coloured.

Cellular content is not of diagnostic value to any extent.

A. B.

[53] **Contribution to the study of Türck's direct pyramidal tract** (Contribution à l'étude du faisceau pyramidal direct de Türck).—J. Nicolesco and T. Hornet. *L'Encéphale*, 1933, **27**, 10.

This study is based on 14 cases of hemiplegia and paraplegia due to cortical or subcortical lesions. The authors group these in six categories: (1) hemiplegia affecting chiefly the upper limb in which there is degeneration of the direct pyramidal tract; (2) paraplegias from cerebral lesions, and (3) hemiplegias affecting the leg principally, each with no degeneration of Türck's tract; (4) hemiplegia with motor loss preponderating in the upper limb, and (5) more complete hemiplegia, each without degeneration of Türck's tract; (6) hemiplegia affecting chiefly the lower limb with degeneration of Türck's tract. The authors support the view that Türck's bundle, being motor to the upper limbs and upper part of the trunk, is most likely to be affected from lesions of the middle part of the ascending frontal convolution or the genu of the internal capsule. This is supported by the cases which they have examined, as most of these fall within the first three categories, the last three including isolated cases which do not follow the general rule. They find no evidence to support the view that Türck's bundle is an extrapyramidal pathway. In fact, a study of its phylogeny indicates that it is of later origin than the crossed pyramidal tract, being associated with the assumption of

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the erect posture by man and the primates, and probably with the more skilful movements of the upper limbs which have been developed with that posture. They have made no attempt to decide whether the fibres of this tract decussate in the cord, but are of opinion that some decussation at least takes place, probably after the fibres have lost their myelin sheaths.

J. G. G.

[54] **The influence of the vestibular nuclei and in particular those of Deiters on tendon reflexes** (Étude de l'influence des noyaux vestibulaires et en particulier des noyaux de Deiters sur la réflectivité tendineuse).—R. Bruynoghe and A. P. Dustin. *Arch. internat. de méd. exp.*, 1932, **7**, 1.

In the decerebrate cat destruction of Deiters' nucleus or interruption of the deiterospinal tract is followed by hypotonia and a change in tendon reflexes, which take the form of those of the spinal animal. This alteration in myotatic reflectivity is the consequence solely of lesions of Deiters' nucleus and its efferent path. The change in the myographic curve results from abolition of muscle tonus and the consequent alteration in length of muscle fibres participating in the reflex.

In man, loss of tendon reflexes may ensue on lesions or functional changes of Deiters' nucleus. In subtentorial tumours loss of the reflexes points to pontobulbar invasion and is an ominous sign.

J. S. P.

[55] **Intracranial hydrodynamics. II. Influence of rapid decompression of the ventriculosubarachnoid spaces on the occurrence of œdema of the brain.**—Jules H. Masserman and Walter F. Schaller. *Arch. of Neurol. and Psychiat.*, 1933, **30**, 107.

The present report is devoted to a description of certain experiments designed to permit direct investigation of the relationship of rapid decompression of intracranial hypertension to the subsequent occurrence of œdema of the brain in dogs. Evidence is presented that cerebral cellular damage may be produced in this way, and this concept, if clinically applicable, may well explain such heretofore empirical tenets as: (1) the necessity of gradual decompression when cerebrospinal fluid is withdrawn by rachicentesis in cases of cranial hypertension; (2) the occurrence of postpuncture cerebral or bulbar symptoms if this precaution is neglected; and (3) the advisability of even maintaining the ventriculosubarachnoid pressure during the intrathecal or intraventricular injection of air for roentgenographic purposes.

R. M. S.

[56] **The mode and site of action of strychnine in the nervous system.**—J. G. Dusser de Barenne. *Physiol. Reviews*, 1933, **13**, 325.

As a generalization, it may be said that strychnine 'takes away all resistances in the various reflex arcs'; its typical tetanic attacks are of central origin, and spinal. Professor Dusser de Barenne concludes from personal experimentation and for other reasons that the alkaloid acts on the perikarya (cell-bodies) in the dorsal horns of the cord, and that tetanic spasms occur solely when these are affected together with the cell-bodies of the motor cells in the ventral grey matter of the cord.

J. S. P.

[57] **The histopathogenesis of primary systematized nervous diseases** (Die Histopathogenese der primär-systematischen Nervenkrankheiten).—Karl Schaffer. *Archiv f. Psychiat.*, 1932, **98**, 130.

Primary systematized nervous diseases can be distinguished from others by a number of different histopathological findings. These diseases begin in the nerve-cells of the centres concerned (e.g. cortical motor centres); they are 'centrogenic,' practically always symmetrical, and in the case of motor diseases the changes begin at the periphery and creep up the systems to their cellular origins. Such diseases are always congenital, in the sense of being due to some inherent, endogenous, defect; they are examples of 'Zentrumabiogenesis.' This is followed by secondary degeneration throughout the systems affected—a 'Systemabiotrophie.' The processes are 'neurogenic,' by which is meant that they involve nerve-cells from within, and are to be clearly separated from 'neurotropic' processes, where the cells are invaded from without. Further, the processes are restricted to neural parenchyma, to neuroectodermal elements. The purely segmental, systematized character of such affections may, however, be partly masked by incidental mesodermal changes.

S. A. K. W.

[58] **Experimental toxic encephalomyelopathy** (Diffuse sclerosis following subcutaneous injections of potassium cyanide).—A. Ferraro. *Psychiat. Quarterly*, 1933, **7**, 267.

Experiments conducted by Dr. Ferraro prove conclusively that a condition of diffuse encephalomyelopathy possessing the histological characteristics of diffuse sclerosis can follow the administration of repeated doses of a toxic substance. He has been able to produce definite processes of demyelination, with typical involvement of axis-cylinders and typical glia reactions; the lesions have shown a predilection for white matter and been more severe in perivascular areas. As regards the identity or otherwise of multiple and of

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diffuse sclerosis Dr. Ferraro ranges himself alongside those who hold that the difference is only one of extension and of intensity, and that the two processes are fundamentally the same. In both multiple and diffuse sclerosis areas of softening and necrosis have been seen, and he has produced them also by his experiments.

Whether his lesions should be considered inflammatory or degenerative is next discussed. In 16 out of 18 cases studied they were purely degenerative, but this statement cannot be made of the other two, and Dr. Ferraro is rather at a loss to explain them. He affirms, however, that under certain circumstances a 'true type' of inflammation may be produced by an aseptic condition or by toxic factors irrespective of a definite infection. This applies in particular to diffuse sclerosis, where an inflammatory, a degenerative, and perhaps a blastomatotic form can be distinguished.

Attention is also directed to the production of areas of demyelination in animals by diets deficient in some vitamins.

J. V.

[59] **Disorders of lipoid metabolism in hereditary nervous diseases** (Störungen des Lipoidstoffwechsels bei Erbkrankheiten des Nervensystems).—W. Spielmeyer. *Klin. Woch.*, 1933, **12**, 1273.

Taking amaurotic family idiocy as his chief topic, Professor Spielmeyer outlines various arguments which in his view prove that the origin of the cellular changes in that affection is the deposition of lipoids in the cytoplasm. Technically, the process is known as 'phosphatid-cellular lipoidosis.' Evidence is also adduced which in his opinion favours the contention that the underlying disorder of the Niemann-Pick affection is a disturbance of lipoid metabolism and that it acts also on the nervous system. Thus there is the same principle of morbid activity in both amaurotic family idiocy and splenohepatomegaly, though the two are not identical. Professor Spielmeyer criticizes the views of Schaffer and allies himself with those who think them untenable.

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[60] **Changes in the central nervous system in smallpox** (Über die Veränderungen im Zentralnervensystem bei Pocken (Variola)).—B. Brouwer, C. L. de Jongh, and R. R. Rochat. *Deut. Zeits. f. Nervenheilk.*, 1933, **131**, 1.

Clinically, the case here described was rather unusual in that the patient lived for some time after the commencement of the illness, thus making it possible for early gliosis to appear as a sequel to the more acute part of the

infection. In consequence, a fair amount of hyperplasia of fibrillar glia was seen in the central nervous system. Otherwise the findings were those of a myeloencephalitis. Their resemblance to the histological picture of vaccine-encephalomyelitis is duly emphasized.

It is gratifying to find that the authors make a stand against the habit of putting affections into one etiological group because of their morphological similarities or identities, on the assumption that the characteristic histological changes result from activation of a latent virus.

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[61] **Malignant tumours of the hypophysis invading the diencephalon.**—E. B. Fink. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 561.

Four cases of malignant tumour of the hypophysis are reported, with post-mortem findings. They all invaded the third ventricle extensively, and some of them involved the cerebrum as far as the frontal lobes in front and the pons behind.

The etiology and pathology of malignant tumours of the hypophysis are reviewed.

The simplest division of malignant tumours of the hypophysis is into adenocarcinoma, for those composed of epithelial elements of the anterior lobe; and craniopharyngeal epithelioma, for the embryonal tumours derived from Rathke's pouch.

The term adamantinoma should be limited to a special class of tumour arising in the tooth germ and occurring in the jaw. True adamantinomas arise in structures which are fully formed and functioning, while embryonal tumours of the hypothesis arise in primitive vestigial elements.

Malignant tumours of the hypophysis composed of epithelial elements of the anterior lobe are uncommon, but they are not exceedingly rare. Malignant tumours derived from remnants of Rathke's pouch are more frequent, and probably all solid tumours of this type are potentially malignant because of their tendency to invade the diencephalon.

Pituitary symptoms are not prominent in malignant tumours of the hypophysis, their occurrence depending somewhat upon whether the growth originates within the sella or is suprasellar from the beginning.

Clinical criteria by which malignancy may be diagnosed are emphasized. The most reliable of these is early and rapidly progressing damage to vision, together with evidence of involvement of the diencephalon. Signs of increased intracranial pressure are late manifestations.

Symptoms indicating invasion of the diencephalon are obesity, polyuria, and hypersomnolence.

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[62] **Chemical studies in the epileptic syndrome.**—H. HOPKINS. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 601.

THE cholesterol content of the blood in patients presenting the epileptic syndrome appears to have some significance in indicating the direction in which physicochemical changes are taking place.

The average whole-blood cholesterol values are slightly lower in a group of epileptic patients than in a corresponding normal group.

The range of variation in whole-blood cholesterol values from hour to hour throughout 24-hour periods is greater in epileptic patients than in normal individuals.

One case undergoing convulsive activity during the 24-hour period of study showed a preparoxymal fall in the whole-blood cholesterol amounting to 11 per cent. of the average cholesterol value for the day.

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SENSORIMOTOR NEUROLOGY

[63] **Clinical and anatomical contributions to the problem of genuine epilepsy** (Klinische und anatomische Beiträge zur Frage der genuinen Epilepsie).—L. v. MEDUNA. *Deut. Zeits. f. Nervenheilk.*, 1933, **129**, 17.

BY the term 'genuine epilepsy' Dr. Meduna appears to mean those cases in which he can find no pathological changes except swollen ganglion-cells with large, pale nuclei. He alleges that 'genuine epilepsy' may begin with Jacksonian attacks, also that Jacksonian and general fits are interchangeable in that condition; and that 'genuine epilepsy' is a chronic disease of the whole central and vegetative nervous systems, whose first sign may be psychical degeneration before fits ever develop. We are informed that the 'characteristic' features of 'genuine epilepsy' are: psychical degeneration, epileptic attacks, and 'pure parenchymatous degeneration' of the brain. But the evidence offered for the validity of Dr. Meduna's claims is rather dubious.

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[64] **Modern ideas on the convulsive state** (Concezione moderna dello stato convulsivo).—M. OSNATO. *Riv. di pat. nerv. e ment.*, 1932, **40**, 362.

THE literature is examined and the results of the author's own work are given. He considers that the changes described by Spielmeyer as occurring in the brains of those subject to convulsive seizures and reproduced experimentally by Gildyea and Cobb represent the final results of a chain of phenomena which constitute the convulsive state.

In all cases there exists something which produces an irritability of the

brain. The lowering of the threshold of response and the sensitization may be produced by a trauma—toxic, infective, or mechanical. The traumata and infections of early childhood form a point for the extension of the epileptogenous area. This alone will not produce an attack, which may be associated with an alteration in the permeability of the vessels caused by trauma, as has been done in experimental work. If the etiological factor is not an illness or traumatic lesion, any disturbance of metabolism causing an increase of lactic acid will produce the same result. These factors alone will not produce a repetition of the convulsion, which depends on the arrival in the cerebral parenchyma of a polypeptid or similar body, which causes an occlusion of the cerebral vessels. This occlusion leads to the changes noted by Spielmeyer and others and to progressive deterioration of the mentality of the epileptic patient. The factors of increased intracranial pressure or asphyxia probably act by increasing the permeability of the vessels, either directly or by means of the autonomic nervous system. They alone cannot produce convulsions, but require the intervention of a ' convulsive agent,' the nature of which must be determined by future research.

R. G. G.

[65] **A malignant meningioma in the left pretemporal region with a ' reactive glioma '** (Meningioma maligno pre-temporale sinistro con glioma reattivo).—S. Gullotta. *Riv. di pat. nerv. e ment.*, 1933, **41**, 38.

A woman of 53 had suffered for five years from signs of increased intracranial pressure and epileptic attacks. She showed left-sided deafness and right-sided diminution of hearing, anosmia and blindness from secondary optic atrophy, giddiness in the erect attitude, Kernig's sign, slight rigidity and tremor of the right arm and a grasp reflex in the right hand. After a short residence in the clinic, she had a stroke and died. Autopsy disclosed a hæmorrhagic focus on the convexity of the left frontal pole, a meningioma in the left pretemporal region and an enlargement of the whole temporal lobe. The invasion of the lobe by the malignant meningioma cells had resulted in a reactive gliomatosis which merits the name of ' reactive glioma ' as suggested by Merzbacher.

R. G. G.

[66] **Syndrome of the infundibulum and tuber cinereum with mental disturbances due to suprasellar tumour** (Syndrome infundibulo-tubérien avec troubles mentaux complexes par tumeur supra-sellaire du troisième ventricule).—A. Obregia, A. Dimolesco and S. Constantinesco. *L'Encéphale*, 1932, **27**, 93.

The case here described appears to have been one of tumour of Rathke's pouch, which began to cause symptoms at the age of 53. In addition to the

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diffuse sclerosis Dr. Ferraro ranges himself alongside those who hold that the difference is only one of extension and of intensity, and that the two processes are fundamentally the same. In both multiple and diffuse sclerosis areas of softening and necrosis have been seen, and he has produced them also by his experiments.

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THE literature is examined and the results of the author's own work are given. He considers that the changes described by Spielmeyer as occurring in the brains of those subject to convulsive seizures and reproduced experimentally by Gildyea and Cobb represent the final results of a chain of phenomena which constitute the convulsive state.

In all cases there exists something which produces an irritability of the

brain. The lowering of the threshold of response and the sensitization may be produced by a trauma—toxic, infective, or mechanical. The traumata and infections of early childhood form a point for the extension of the epileptogenous area. This alone will not produce an attack, which may be associated with an alteration in the permeability of the vessels caused by trauma, as has been done in experimental work. If the etiological factor is not an illness or traumatic lesion, any disturbance of metabolism causing an increase of lactic acid will produce the same result. These factors alone will not produce a repetition of the convulsion, which depends on the arrival in the cerebral parenchyma of a polypeptid or similar body, which causes an occlusion of the cerebral vessels. This occlusion leads to the changes noted by Spielmeyer and others and to progressive deterioration of the mentality of the epileptic patient. The factors of increased intracranial pressure or asphyxia probably act by increasing the permeability of the vessels, either directly or by means of the autonomic nervous system. They alone cannot produce convulsions, but require the intervention of a ' convulsive agent,' the nature of which must be determined by future research.

R. G. G.

[65] **A malignant meningioma in the left pretemporal region with a ' reactive glioma '** (Meningioma maligno pre-temporale sinistro con glioma reattivo).—S. Gullotta. *Riv. di pat. nerv. e ment.*, 1933, **41**, 38.

A woman of 53 had suffered for five years from signs of increased intracranial pressure and epileptic attacks. She showed left-sided deafness and right-sided diminution of hearing, anosmia and blindness from secondary optic atrophy, giddiness in the erect attitude, Kernig's sign, slight rigidity and tremor of the right arm and a grasp reflex in the right hand. After a short residence in the clinic, she had a stroke and died. Autopsy disclosed a hæmorrhagic focus on the convexity of the left frontal pole, a meningioma in the left pretemporal region and an enlargement of the whole temporal lobe. The invasion of the lobe by the malignant meningioma cells had resulted in a reactive gliomatosis which merits the name of ' reactive glioma ' as suggested by Merzbacher.

R. G. G.

[66] **Syndrome of the infundibulum and tuber cinereum with mental disturbances due to suprasellar tumour** (Syndrome infundibulo-tubérien avec troubles mentaux complexes par tumeur supra-sellaire du troisième ventricule).—A. Obregia, A. Dimolesco and S. Constantinesco. *L'Encéphale*, 1932, **27**, 93.

The case here described appears to have been one of tumour of Rathke's pouch, which began to cause symptoms at the age of 53. In addition to the

bitemporal hemianopia with optic atrophy which is usual with tumours of this kind, mental symptoms, chiefly confabulation, and somnolence were very marked. There was in addition glycosuria, a certain degree of polyuria, boulimia and, towards the end, hypothermia. The tumour, which was irregularly cystic, lay between the hypophysis and the infundibulum. It compressed the optic nerves and chiasma above it, indicating its origin fairly low in the infundibular stalk. Examination of the hypothalamic nuclei showed some atrophy of nerve-cells and many degenerated cells undergoing satellitosis. There was also some perivascular infiltration with lymphocytes and macrophages in this region. Small ' nodules inflammatoires ' were seen in the brain tissue just under the capsule of the tumour. There was also some degeneration of nerve-cells in the deeper layers of the cortex, most marked in the frontal and occipital regions, but no perivascular cuffing was seen here. Whether the mental state had any relationship with the tumour is by no means clear. The case is not otherwise of a very unusual nature.

J. G. G.

[67] **The anatomical cerebral substratum for the faculty of arithmetical calculation** (Über das hirnanatomische Substrät des Rechentalentes). —K. v. Santha. *Archiv f. Psychiat.*, 1932, **98**, 313.

This communication cannot well be summarized since it is replete with discussions of interest as regards arithmetical talent or genius, right- and left-brainedness, mental inequality, the relation of genius to psychopathy, and kindred matters.

Briefly, the case is that of a man who was known as a ' Rechenskunstler.' His family history was not good, and he himself had periods of mental abnormality, necessitating his admission to an institution. From early years he had been remarkable for his faculty of reckoning, but he was no mathematician. As a little boy of eight he was able to make calculations ' in his head,' dealing with numbers of five or six figures in all sorts of ways; he could give day of the week for any given date in month and year, and so on ; numerous examples are cited. His memory was phenomenal in this respect, for he had devised schemes of his own to make his calculations seem almost instantaneous. Otherwise he was mentally undistinguished.

At autopsy the brain was found to be considerably below average weight (1185 g.). The chief morphological feature was the rich convolutional pattern in respect mainly of the region of the angular gyrus—more so on the right than on the left, though the patient was right-handed. Histologically, the fourth or internal granular layer was bigger than normal.

S. A. K. W.

[68] **An additional case of hæmangioblastoma of the retina and cerebellum, with a note on Lindau's disease.**—H. R. Viets. *Jour. Nerv. Ment. Dis.*, 1933. **77,** 457.

A case of Lindau's syndrome is reported in a man 48 years of age. Although there were some symptoms, with blurring of vision, in the right eye for four years, this has not interfered with the patient's occupation. Signs of cerebellar dysfunction with increased intracranial pressure developed rapidly a few weeks before he was seen. On entrance to hospital von Hippel's disease of the retina was recognized, and cerebellar exploration was done at once with removal of cystic fluid. The cerebellar symptoms disappeared after the operation and the patient remained well ten months later. The retinal changes, however, have continued to advance and will probably entail enucleation of the eye. Only about 40 cases of the complete syndrome of retinal and cerebellar hæmangioblastoma have been described in the literature, a condition which is now well recognized.

R. G. G.

[69] **Hæmangioma cavernosum of thalamus and midbrain** (Über einem Fall von Hæmangioma cavernosum im Sehhügel und Mittelhirn).—B. Hechst. *Zeits. f.d.g. Neurol. u. Psychiat.*, 1932, **142,** 590.

In this case a cavernous hæmangioma was found in the upper part of the third ventricle, invading the mesial aspects of both optic thalami posteriorly, and spreading to the anterior corpora quadrigemina, etc. Among the clinical symptoms of a general kind were loss of spontaneity, of impulse to action, intense sleepiness, cataleptic phenomena, immobility, amimia, and others also of a psychopathological kind. With the tumour hydrocephalus co-existed, while in both blood and fluid the Wassermann reaction was positive.

Despite this pathological complexity, Dr. Hechst seems to believe his case justifies the conclusion that the lesions of the optic thalamus explain the catatonia and other 'psychomotor' symptoms which were observed during life.

S. A. K. W.

[70] **Hallucinations associated with meningiomas of the small wing of the sphenoid** (Hallucinations et méningiomes de la petite aile du sphénoïde).—M. David and P. Puech. *L'Encéphale*, 1932, **27,** 409.

It is remarkable that Hughlings Jackson's uncinate fits or 'dreamy states' should occur much more frequently with gliomata than with meningeal tumours. For whereas uncinate fits occurred in 40 per cent. of Cushing's cases of glioma of the temporal lobe, the two cases reported here have been the only ones presenting this symptom in the clinic of Clovis Vincent among

25 cases of meningioma of the small wing of the sphenoid. In both cases uncinate fits with or without loss of consciousness were the first symptom and were prominent throughout the course of the illness. The side of the tumour, which in both cases was the right, was indicated by X-rays, which showed some erosion of the corresponding wall of the sella turcica and of the lesser wing of the sphenoid. In one case, however, ventriculography had to be practised before the diagnosis was certain.

J. G. G.

[71] Xanthomatosis and the central nervous system (Schüller-Christian syndrome).—CHARLES DAVISON. *Arch. of Neurol. and Psychiat.*, 1933, **30**, 75.

XANTHOMATOSIS, the Schüller-Christian syndrome, generally characterized clinically by the symptom triad of defects in the membranous bones, exophthalmos and diabetes insipidus, has as its histological basis a diffuse collection of lipoid cells ('foam cells'), cellular reactions and fibrosis in various organs of the body. The granulomatous collections within the skull and dura mater giving rise to neurological symptoms have been described frequently. The polyuria and polydipsia are believed to result from pressure on the hypophysis and tuber cinereum or from direct invasion of these structures by such lipoid masses.

In the case reported by the author, the clinical picture of the disease process, unlike many of the cases, started when the patient was 22, with pain and falling out of the teeth. Deposition of granulomatous tissue in various organs soon followed, accompanied by symptoms referable to these organs. Three years later, diabetes insipidus was first noticed. At the age of 28, a few neurological symptoms and signs appeared—horizontal nystagmoid twitches and loss of taste over the left anterior two-thirds of the tongue. The neurological signs progressed and, two years after their appearance, there developed involvement of the pyramidal tracts, cerebellum, left sensory fifth nerve and left peripheral facial nerve, and slight involvement of the eighth nerve. The cholesterol content of the blood serum was normal; the total fat was increased.

As in most cases of Schüller-Christian disease, the bony changes were not confined to the skull, but were found also in other parts of the skeletal system. These changes as demonstrated were predominantly osteoclastic, although evidence of osteoplastic changes were also reported.

In addition to the deposits of lipoid cells found in most of the organs, the white matter of the central nervous system was the seat of numerous demyelinated plaques filled with compound granular corpuscles and giant glia cells. These two types of cells found in the neural structures are considered analogous to the foam cells and the reactive type of cells (fibrosis and inflammatory cells) demonstrated in other organs. In this disease the hypo-

physis was the seat of deposits of foam cells, while the tuber cinereum region showed evidences of reactive phenomena (gliosis, fibrosis and inflammatory cells) and occasional compound granular corpuscles and giant glia cells. The author concludes that the process is one of disturbance in lipoid metabolism (cholesterol), and in some respects resembles Gaucher's and Niemann-Pick's diseases, and possibly amaurotic family idiocy.

R. M. S.

[72] **Abrupt slowing of the cerebral circulation.**—T. G. Inman. *Calif. and West. Med.*, 1932, **36**, 1.

In a review of the histories of a number of patients with arteriosclerosis in whom transient cerebral symptoms frequently occurred, the author noted that the attacks often followed a change to the upright position. The greatest number came on in the morning on getting up, or shortly afterwards; in others the attack came when fatigue was evident. Among younger persons he also noticed cases where giddiness and fainting occurred when they were standing quietly. In both classes observation showed a systolic blood-pressure lower than normal, or a greater fall than normal on the assumption of the erect posture. His theory is not one of cerebral anæmia, but one attributing the syndrome to more or less abrupt slowing of the cerebral circulation, carried to a degree at which a localized area of the brain is temporarily deprived of its customary amount of arterial blood. With this slowing there is passive congestion of brain capillaries, both arterial and venous. In the presence of myocardial weakness, loss of vasomotor tone, inadequate support of the splanchnic circulation, or generalized muscular weakness, external vessels may not compensate sufficiently for the immediate needs of the internal vessels, hence the capillary stasis is not overcome. The situation is one in which thrombosis may and doubtless does occur, but if the vessel intima is healthy it may not develop. Persons with relaxed abdominal musculature and poorly compensating splanchic vasomotor systems are specially liable to suffer in this fashion.

J. V.

[73] **The late commotional syndrome of closed cranial wounds** (Le syndrome commotionnel tardif dans les traumatismes fermés du crâne).—F. Bremer, G. Hicguet, H. Coppez, and P. Martin. *Revue d'oto-neuro-ophtalmol.*, 1932, **10**, 161.

This is a long and comprehensive review of the salient features of the syndrome which often follows, after a period, different kinds of closed head wound. The facts are set forth seriatim from clinical, pathological, and pathogenic standpoints. The paper is well worth the attention of the neurologist who has to deal with medicolegal cases.

Subjective symptoms include headache, giddiness, nervousness, insomnia, defective memory, mental asthenia, retinal asthenia, noises in the ears, genuine vertigo, etc. Objective features comprise exaggerated reflexes, tachycardia, hyperidrosis, thermal instability, and so on. Of some value are the results of investigation of retinal tonoscopy, retinal venous pressure, the oculocardiac reflex, cochlear and vestibular function, the pressure of the cerebrospinal fluid and its composition. There is a full description of recent studies of the pathological anatomy of closed head wounds.

In the opinion of the writers, the syndrome is determined by the following factors :

1. ganglion-cell and neuroglial lesions ;
2. disordered cerebral circulation—in particular, hyperæmia ;
3. changes in cerebrospinal fluid pressure and composition ;
4. the individual psychological reaction of the person concerned.

S. A. K. W.

[74] **Subarachnoid hæmorrhage and its relation to aneurysm** (Über die sogenannten spontanen subarachnoidalen Blutungen und ihre Beziehung zum Aneurysma der Hirngefässe).—A. Biemond and J. W. G. ter Braak. *Deut. Zeits. f. Nervenheilk.*, 1933, **132,** 4.

This useful review and contribution to the subject of subarachnoid hæmorrhage is based on the investigation of some 40 cases which have occurred in the Nerve Clinic at Amsterdam during the last seven years. The clinical features of the condition are discussed at some length ; Korsakow's psychosis is illustrated, and the point made that its presence or absence is without influence on the prognosis. Much attention also is devoted to ocular symptoms and signs, to retinal hæmorrhages and the like. Such hæmorrhages were found in six of the 40 cases. Mention is made also of papillœdema, with or without local hæmorrhages. An interesting annotation refers to the development of pseudo-infective symptoms (fever, leucocytosis in blood, herpes labialis, etc.). Albuminuria was observed with some frequency.

Of the 40 cases, 18 proved fatal. Two patients died later, at home ; of the remainder 10 are known to have continued well. By way of treatment repeated lumbar puncture is recommended.

Sixteen cases were examined post mortem ; in 15 a ruptured basal aneurysm was found.

S. A. K. W.

[75] **The biological diagnosis of cysticercus of the neuraxis** (La diagnosi biologica di cisticercosi del nevrasse).—C. Rizzo. *Riv. di pat. nerv. e ment.*, 1933, **41,** 193.

A fourth case (the others have been noted in previous issues of the *Rivista* and in abstracts in this Journal) of cysticercus diagnosed by the presence of

eosinophils in the cerebrospinal fluid is described. It was that of a man of 61 who suffered for about four months from Jacksonian epilepsy of the facial muscles of the right side and sometimes of the right upper limb. There was a transitory dysarthria after these attacks, and a slight but permanent apraxia of the limbs on the right. The cerebrospinal fluid showed a leucocytosis of 12.5 to 20 per c. mm. at first, rising to 102, of which 10-40 per cent. were eosinophils.

R. G. G.

[76] **Chronic subdural hæmatoma** (Les hématomes sous-duraux chroniques). —P. Van Gehuchten and P. Martin. *Revue neurol.*, 1932, **2**, 178.

Some four cases of chronic subdural hæmatoma are described fully, both clinically and histopathologically. The vexed question of pathogenesis is discussed and an eclectic attitude adopted. The writers affirm that in some examples of the condition the pathological findings support the theory that meningitis first involves the dura and is succeeded by hæmorrhage, but that in the greater number hæmorrhage is primary, resulting from a trauma so slight as almost or actually to pass unperceived. The rarest cases are those of primary bleeding without injury. The organization of the initial hæmatoma itself is such as almost inevitably to give rise to subsequent extravasations.

S. A. K. W.

[77] **On the grasp reflex and motor negativism as symptoms of a focal cerebral lesion** (Sui fenomeni di prensione e di negativismo motorio quali sintomi di lesioni cerebrali a focolaio).—S. Gullotta. *Riv. di pat. nerv. e ment.*, 1933, **41**, 226.

Examples of this syndrome have probably not been recognized as often as they might have been, as the author was able to collect five cases in a year. The commonest phenomenon is a forced grasp which can be voluntarily relaxed. One patient manifested a general tendency to grasp as if there were a sort of ' hyperæsthesia ' to this end. He showed localized motor negativism with slight hypertonia and slight pyramidal signs.

Only in one case was there frontal hæmorrhage, but in all cases, in addition to an internal hydrocephalus, there was a striate and thalamic lesion. The author concludes that this grasp reflex may occur normally under violent emotion ; when it occurs pathologically, if it is bilateral and inconstant it indicates internal hydrocephalus. If it is circumscribed or only on one side it has localizing value. If the forced grasp cannot be voluntarily relaxed, or does not relax on painful stimuli, it is indicative of contralateral frontal lesion, especially if accompanied by signs of cerebral tumour. If, however, it can be voluntarily relaxed and is not increased by pain, and is accompanied by motor negativism, it indicates a lesion in the neostriatum and contralateral thalamus,

especially if there are also signs of cerebral arteriosclerosis. Probably there is no one centre which inhibits the forced grasp, the 'centre' for grasping in the cord becoming released by various higher motor lesions. The 'centre' for motor negativism is uncertain, but if the symptom is circumscribed it probably indicates a lesion of the contralateral basal ganglia.

R. G. G.

[78] **The initial stages of the Parkinsonian forms in chronic epidemic encephalitis.**—K. H. KRABBE. *Acta Psychiat. et Neurol.*, 1932, **7**, 317.

DR. KRABBE has studied 60 cases of definite and unmistakable encephalitic Parkinsonism. In six of these the original disease developed in a cryptogenic fashion; the remaining 54 were preceded by an affection which at the time of its appearance was diagnosed as a typical encephalitis, or as influenza. In about one-half of these cases the Parkinsonian syndrome developed in continuation of the acute febrile initial stage, either during it, or soon after, the two then being separated by a period in the course of which tiredness, headache, insomnia, or somnolence, were in evidence. But in the other half of the cases the syndrome did not unfold till after some months or years of symptom-free life.

It seems natural to conclude that the preceding 'influenza' has in reality constituted an attack by the encephalitis virus, and not been an accidental infection by some other virus unrelated to encephalitis.

A. B.

[79] **Oculogyric crises and Parkinsonism in cerebrospinal syphilis** (Crises oculogyres et Parkinsonisme dans la syphilis cérébrospinale).—K. H. KRABBE. *Acta Psychiat. et Neurol.*, 1931, **6**, 457.

FOUR cases of characteristic Parkinsonism and oculogyric crises are described in this article, in two of which there was evidence of old, preceding syphilis, though tests were negative. In the other two, oculogyric crises occurred, and Wassermann tests were positive. The author is evidently inclined to consider these two as examples of ordinary epidemic encephalitis complicated by occult syphilis, but suggests the possibility that Parkinsonism and oculogyric crises may arise on a basis of syphilitic encephalopathy, though no proof is offered. The title of his article is therefore calculated to mislead.

A. B.

[80] **Contribution to the knowledge of rare syndromes due to embryonic tumours** (Contributo alla conoscenza delle sindromi rare da tumori embriogenetici).—P. OTTONELLO. *Riv. di pat. nerv. e ment.*, 1933, **41**, 512.

A YOUNG woman of 20 developed a sensory and motor paraplegia, and in the course of a fortnight's examination showed the clinical signs of a dorsal

lesion and a blocking of the arachnoid space. This suggested an extramedullary teratoma. A dermoid was completely removed at operation and a certain measure of recovery ensued. This is the fifth case described in the literature of the rare spinal distribution of an embryonic tumour.

R. G. G.

[81] **A case of polyganglionitis (poliomyelitis posterior) with the clinical picture of subacute pseudotabes** (Über einem Fall von Polyganglionitis (Poliomyelitis posterior) unter dem klinischen Bild einer Subakuten Pseudotabes).—K. v. Santha. *Archiv f. Psychiat.*, 1933, **100**, 398.

The case described is that of a man of 63, whose clinical symptoms began with motor and sensory impairment in the lower limbs, ataxia, loss of deep reflexes, paræsthesiæ, pains, frequency of micturition. The upper limbs were normal, and Wassermann reactions negative. With only seven cells per cubic millimetre in the fluid, there was a great increase in total protein. Later, his condition became worse; muscular weakness of all limbs developed, and the muscles began to waste. Electrical reactions never showed any abnormality.

Much the most striking feature of the case pathologically was the gross toxidegenerative changes of the dorsal root ganglia and dorsal roots, while the ventral were normal. In addition, there was a tabes-like sclerosis of the dorsal columns of the cord. In the peripheral nerves no neuritis was seen, but much secondary degeneration presumably of sensory as opposed to motor fasciculi. Changes were also found, however, in pons and medulla (descending root of the fifth, nucleus solitarius, etc.).

Histologically, the reactions constituted a mixed ectomesodermal type. The case is considered to be, not one of polyneuritis, but of localized polyganglionitis or posterior poliomyelitis.

S. A. K. W.

[82] **Chronic adhesive spinal pachymeningitis and leptomeningitis following a melitococcal meningitis** (Pachi e leptomeningite cronica spinale adesiva consecutiva a meningite melitococcica).—G. G. Noto. *Riv. di pat. nerv. e ment.*, 1932, **40**, 440.

This complication of Malta fever is described for the first time in a case in which the nervous lesion was a meningitis at the level D 7-8. The case illustrates the differentiation between spinal tumour and obliterative meningitis, and, in the view of the author, also the contraindication for surgical treatment in such cases.

R. G. G.

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[83] **The value and significance of the so-called meningitic signs of Kernig, of Brudzinski, and of hyperalgesic reflexes** (Sul valore e sul significato dei cosidetti segni meningitici di Kernig, di Brudzinski, e dei riflessi iperalgesici).—S. Gullotta. *Riv. sper. di fren.*, 1933, **57**, 1.

In six cases of lesion of the corpus striatum, confirmed anatomically, Kernig's and Brudzinski's signs were present in the lower limb opposite to the side of the lesion. Also, the 'hyperalgesic reflexes' of Babinski and Jarkowski were similarly obtained on stimulating the contralateral lower limb. The unilaterality of the phenomena is relied on as proof that they could not have been due to meningitic involvement, but resulted from the focal cerebral disorder. The view here taken is that the presence of such 'meningitic' signs is secondary to release of tonic centres from higher inhibitory systems.

S. A. K. W.

[84] **'Alcoholic' polyneuritis : dietary deficiency as a factor in its production.** —G. R. Minot, M. B. Strauss, and S. Cobb. *New England Jour. Med.*, 1933, **208**, 1244.

This paper merits more than passing attention, short though it is. From observations on 57 cases during the last five years, and a study of the records of 73 additional cases, the authors conclude that dietary defects, more particularly lack of vitamin B_1, play a significant part in the development of 'alcoholic' neuritis. Not only is there generally an inadequate intake of food for some time previously, but also the state of the gastrointestinal tract and the presence of factors inhibiting the effectiveness of nutritional elements have to be borne in mind. Diets rich in complete proteins, minerals, and vitamins have been administered, and the authors affirm that under this regime recovery has been observed more uniformly and with greater rapidity than before.

J. V.

[85] **The central paths and receptors for pain** (Contribucion anatomo-clinica y quirurgica a las vias y receptores centrales del dolor).—V. Dimitri and M. Balado.

The reprint supplied by the authors does not indicate in what journal this paper was published. It is of considerable interest.

The case was that of a man of 55, who developed suddenly a fairly complete right hemiplegia, and this was soon followed by severe and progressively acute 'central' pains on the right side. Though the motor weakness improved the pains became worse, and were associated with choreoathetoid attitudes and movements of the affected limbs. Tactile sensibility was little affected, whereas both painful and thermal appreciation was much impaired, also deep sensibility.

In view of the severity of the pains, the left parietal cortex was exposed and the whole of the middle and a large part of the foot of the ascending parietal convolution and of the convolution behind it were extirpated by the method of electrocoagulation. This procedure was without effect on the pain, and accordingly about six weeks later the corpus callosum was exposed and incised in an anteroposterior direction. From this operation the patient did not recover.

At the post mortem the lesion was found to consist of an area of softening in the left hemisphere, which was situated behind the lenticular nucleus, and which involved, inter alia, striothalamic and thalamocortical fibres, accounting for the clinical symptoms sufficiently well. The lesion was not in the thalamus itself, and its situation shows that a thalamic syndrome need not be attributed solely to invasion of the thalamothalamic system. Further, the entering centripetal sensory thalamic fibres were preserved, whereas those between thalamus and cortex were affected. The authors affirm that since cellular thalamic changes were purely secondary to retrograde degeneration from this lesion of the thalamocortical system they could not be the cause of the pains. It is also suggested that since extirpation of the ascending parietal did not relieve the pains the cortical centre for their appreciation must be situated elsewhere.

S. A. K. W.

[86] **Pyramido-extrapyramidal hemiplegia** (Hemiplejia piramido-extrapiramidal).—V. DIMITRI. *La semana medica*, 1931, **38,** 1.

THE case described, that of a man of 47, syphilitic, was one of right hemiplegia and motor aphasia, coupled with athetosis, loss of sensibility in the affected arm, astereognosis, etc. Post mortem a large softening was found to extend over the cortex in the distribution of the anterior part of the left sylvian artery, together with softening of the corpus striatum and thalamus in various degrees at various levels. In view of the enormous size of the lesions it is difficult to correlate the clinical phenomena with specific pathological findings. The type dignified by the author as pyramido-extrapyramidal hemiplegia is far from uncommon. He considers, inter alia, that the absence of the Babinski plantar reflex in his case is due to concomitant extrapyramidal symptoms, and also argues that the degeneration of the anterior horn cells found on the hemiplegic side is caused by failure of trophic influence exercised thereon by the rubrospinal tract.

S. A. K. W.

[87] **Muscular rigidity in arteriosclerotics** (Contribution à l'étude de la rigidité musculaire des artério-scléreux).—A. KREINDLER and A. BRUCH. *L'Encéphale*, 1932, **27,** 224.

THE case here reported is of special interest in that paraplegia in flexion with generalized muscular rigidity of an extrapyramidal type appeared to be due

to vascular lesions in the basal ganglia. The patient was an old man of 78 who had had slight strokes with temporary right hemiplegia nine years, and again seven years, before death. For the last four years he had become generally weaker physically and mentally, and had been bedridden for two years. After a further slight stroke about 18 months before examination his lower limbs as well as his arms became fixed in flexion. Voluntary movements were possible but very slow, and did not produce relaxation either in the flexors or extensors of the limb, and there was extreme hypertonicity of the cogwheel type. The tendon reflexes were present, the abdominals feeble, the right plantar was flexor and the left equivocal. After injection of half a milligram of scopolamine the muscular rigidity was greatly reduced and ankle clonus and a definite Babinski response appeared on the right side. The chronaxy of the limb musculature was generally increased, but much more in the muscles than in the nerves, so that there was some 'heterochronism' between these. After scopolamine, however, the chronaxy of the muscles was decreased and corresponded more closely with that of the nerves.

Post-mortem examination showed considerable atheromatous changes in the cerebral vessels. There was a large softening in the centre of the right lenticular nucleus, destroying most of the globus pallidus and part of the putamen. Smaller areas of softening were scattered through the corpus striatum and the internal and external capsules on both sides, and there was a definite 'état criblé' in the basal ganglia. Apart from a small softening in the right lateral nucleus the thalamus was spared on both sides. A small softening was also present in the left ventral part of the pons. There were some changes in the nerve-cells of the substantia nigra and red nucleus but no gross softening in the midbrain.

On such widespread lesions it is, as the authors recognize, impossible to base any theory as to the causation of the unusual form of paraplegia found in this patient. But they hope that the collection of similar cases may throw some light on this problem.

J. G. G.

[88] **The costopectoral reflex** (Le reflesso costo-pectorale : suo valore semeiologico nelle sindromi di compressione midollare).—G. G. Noto. *Riv. di pat. nerv. e ment.*, 1933, **40**, 477.

When the seventh rib is percussed at a point or zone comprised between the anterior and mid-axillary lines the lowermost fasciculi of the pectoralis major can be seen to contract. Dr. Noto is of the opinion that this reflex has localizing value ; its afferent arc is the seventh thoracic root, whence the impulse passes up in the cord to the level of the eighth cervical and first thoracic ; here the efferent arc leaves the cord to proceed to the pectoralis. In two cases of spinal tumour with compression at the level of the third and fourth thoracic segments the reflex was abolished.

J. V.

[89] **The reflex of Rossolimo** (Recherches sur le réflexe de Rossolimo).—K. H. KRABBE and M. ELLERMANN. *Acta Psychiat. et Neurol.*, 1933, **8**, 241.

AN examination of Rossolimo's reflex in some 150 cases of various kinds of nervous disease disclosed its presence in 32. In 27 of these, a Babinski plantar response coexisted. Among 119 cases, however, where its absence was noted, a Babinski response occurred in 53. The Rossolimo sign is frequent but not constant in disseminated sclerosis. It has no pathognomonic value and no special significance beyond its occasional presence in early cases of pyramidal disorder without either exaggerated deep reflexes or Babinski reflex.

J. V.

[90] **Clinical observations on the value of the Hoffmann sign.**—T. FAY and H. B. GOTTEN. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 594.

THE Hoffmann sign is elicited as follows : the hand of the subject is grasped, so that the ulnar portion of his palm rests in the examiner's hand, his forearm in the mid-position between pronation and supination, his fingers relaxed. His attention is then diverted. The examiner snaps with his thumb the nail of the middle or ring finger of the subject. Slight or no movement of the thumb and index finger of the subject indicates that no organic nervous disease is present above the fifth cervical segment of the cord or involving the brain surface and cortical spinal pathways. If such a lesion does exist, however, a quick involuntary approximation of the thumb and index fingers will follow. Movement of the thumb or index finger alone cannot be regarded as a positive response, but simultaneous flexion of each in a movement of approximation is considered positive, and has been found as reliable as the Babinski reflex when proper testing has been carried out. This sign appears to be a delicate reflex phenomenon associated with organic disease of the nervous system situated above the mid-cervical region. It compares favourably with the Babinski reflex as a reliable index of organic disease in the cortical spinal pathways. It may at times appear without a Babinski sign when the lesion is focal to the fibres or areas concerned in motor function of the upper extremities.

The results of these studies have convinced the authors that the Hoffmann reflex is valuable and reliable and should find wider application in routine clinical examinations.

R. G. G.

[91] **Tympanic plexus neuralgia.**—F. L. REICHERT. *Jour. Amer. Med. Assoc.*, 1933, **100**, 1744.

MORE than 40 cases are known in the literature of ordinary glossopharyngeal neuralgia, characterized by paroxysmal attacks of lancinating pain starting in

the tonsillar region or base of the tongue and frequently radiating to the ear, often accompanied by salivation. Such attacks are generally induced by eating, talking, or swallowing, or by other movements of pharynx and tongue.

Dr. Reichert describes a rare variety of partial involvement of the ninth—a tic douloureux of the tympanic branch (Jacobson's), wrongly attributed, in his opinion, to the sensory filaments of the seventh (geniculate neuralgia). It is distinguished by paroxysms of stabbing pain in the external auditory meatus, often associated with other pains in face and postauricular region, not induced by talking, eating or swallowing, and not attended by salivation.

It can be cured by intracranial division of the glossopharyngeal nerve.

J. V.

[92] **On the transmission of Huntington's chorea for 300 years : The Bures family group.**—P. R. Vessie. *Jour. Nerv. Ment. Dis.*, 1932, **67**, 553.

Huntington's chorea is transmitted directly and persistently from generation to generation. The relentless intelligence which controls this faulty evolution shows no disposition to revoke or alter the nature of the severe heritage.

Pathologists report extensive, multifarious lesions in the brain structures, but agree in final analyses on constant atrophic changes in the corpus striatum and cerebral cortex. It is not possible to state with any certitude where the initial lesions begin.

The release of the uncontrollable, bizarre movements has been interpreted as an overactive influence from the globus pallidus type of large cells, which fail in their automatic and associated responses, because the inhibitory function of the small type of neostriatal cells has been destroyed by an elective atrophy. The similar process of atrophic degeneration in the cortex and subcortex may implicate complexly the system of motor pathways.

A congenital lack of vitality in the nervous system is evidenced by irritability in the personality make-up of many members destined to this type of chorea.

The early psychosis in the condition manifests frequently a psychological background as a result of irritability, sensitiveness, fear, anxiety states, suspicion, shame and social ostracism. Memory impairment, deterioration of interests and other gross mental changes are the result of cortical pathology.

The author has ascertained, through an independent study of the earliest colonial records, that the group of ancestors which contributed largely to hereditary chorea, had emigrated from Bures, Suffolk, in 1630. It is his opinion that the common stock comprised principally three men and their wives. The three men were illiterate and arrogant, and none attained recognition or respectability. Not only do the archives disclose behaviour problems in these men, but their wives are suspected of being the means of transporting a family disease from England to the colonial states, which

inheritance has spread throughout the United States. The author further believes the true story of this lesion to be revealed in the witchcraft trials of women in the Bures group.

R. G. G.

PROGNOSIS AND TREATMENT

[93] **Surgical relief of the headache of migraine.**—D. G. Dickerson. *Jour. Nerv. Ment. Dis.*, 1933, **77**, 42.

Various theories bearing upon the etiology and pathology of migraine are discussed, and although the numerous therapeutic measures that have been advocated are not taken up in detail, they are briefly alluded to. Seven cases of patients suffering from headache diagnosed as migraine, treated by ligation and section of the middle meningeal artery, are briefly presented.

While these few cases do not absolutely prove that the headache of migraine can be relieved by ligation of the middle meningeal artery, the author hopes that investigators will follow out this procedure in similar cases which have not been benefited by the customary line of treatment.

R. G. G.

Psychopathology

PSYCHOLOGY

[94] **Pleasure and pain and the emotions.**—M. S. Kruger. *Brit. Jour. Med. Psychol.*, 1933, **13**, 51.

Pleasure and pain are emotions, not sensations in the usual sense. So-called 'physical' pain (pleasure) is any type of sensation which gives rise to the emotion of pain (pleasure), while so-called 'mental' pain (pleasure) is the emotion of pain (pleasure) arising in response to a thought which is not a sensation. Pleasure is the organic welcoming (receiving within) reaction, while pain is the organic shrinking (excluding) reaction. Sensations (which include appetites and emotions) are pleasurable, indifferent or painful according as they do or do not conduce towards the satisfaction of vital needs. Probably all the 'emotional' reactions are in essence the same for all species, being based upon the main types of behaviour possible to any organism. Emotions (including appetites) may be divided into four main groups: those of desire, rejection, satisfaction and non-satisfaction. The position of the hedonists seems to be sound if we understand by pleasure (pain), not the emotion, but the pleasant (painful) sensations or other thoughts which give rise to the emotion, and if we realize that there is fundamentally no difference between instinctive and intelligent action or between function and behaviour.

C. S. R.

[95] **The biologic basis of conflict in behaviour.**—G. E. Coghill. *Psychoanalytic Review*, 1933, **20**, 1.

The purpose of this paper is to show that there are, in the development of behaviour, two essentially antagonistic principles, and that these principles prevail also in the development of the nervous system. It is noted first, that the early behaviour pattern of the individual expands as a primarily integrated unit; secondly, that partial patterns arise by individuation within the integrated total pattern; thirdly, that there is a definite neural mechanism of the total pattern; fourthly, that mechanisms of partial patterns arise secondarily and are essentially antagonistic to the mechanism of the total pattern; fifthly, that the mechanism of the total pattern is a growing organ. Finally, the suggestion is offered that these antagonistic processes may be the organic basis of psychic conflict.

C. S. R.

[96] **What is the psychology of organic epilepsy?**—L. Pierce Clark. *Psychoanalytic Review*, 1933, **20**, 79.

The brain and the body are essential in the ego's task of meeting reality; and where these are injured in any way there is a severe handicap to the personality as a whole. An organic lesion may not only cripple the brain but commonly puts out of action certain parts of the body which are also useful to the ego in carrying out its ordinary functions. The injury is a wound to the ego's sense of well-being and completeness; some form of emotional compensation is necessary. There is the tendency for the ego to impound more libido into the resulting narcissism than is really necessary. Such overcompensation has two harmful results. Firstly, it tends to cramp the individual in the full development of his emotional energy towards adult capacities, and there may be narcissistic fixation. Secondly, because of the failure to discharge libido objectively, there is a steady inbinding of tension within the ego. Epileptic attacks, perhaps originally traceable to the lesion itself, now become exaggerated as a mandatory, automatic discharge of this tension. Moreover, the ego in its weakness is characteristically unable to sustain an aggressive socialized use of the destructive impulses, and these, too, are mainly turned inward. Again the impulsive energy is directed upon the ego—in this instance with a violence which is expended in the convulsion. Much may be done in helping the ego to gain strength and to avoid the excessive inbinding of narcissistic libido.

C. S. R.

NEUROSES AND PSYCHONEUROSES

[97] **Masked thyrotoxicosis simulating primary neurosis.**—S. Ginsburg. *Jour. Nerv. Ment. Dis.*, 1932, **76**, 331.

A study of six cases and a review of the literature lead the author to conclude that,

Thyrotoxicosis is a disease which affects persons of all ages from early infancy to advanced life.

Emotional shock and strain—biological, social-economic, or sexual-traumatic—in apparently normal or neuropathic individuals, may lead to the clinical development of exacerbation of atypical thyrotoxicosis.

Marked thyrotoxicosis may exist for many years without any thyroid enlargement or exophthalmos.

The constitutional symptoms of thyrotoxicosis resemble closely the manifestations of the neuroses and, in the absence of thyroid enlargement and exophthalmos, have been mistaken for pure neuroses.

Many cases of nervous indigestion have been proved to be thyrogenous in origin.

A depressive psychosis may develop in the course of thyrotoxicosis, not as a mere casual phenomenon, but definitely dependent upon toxic thyroid products. Such a psychosis has cleared up and failed to recur when the thyrotoxic condition was controlled.

In cases of neurosis or depressive psychosis associated with tachycardia, tremor, heat intolerance, excessive sweating, loss in weight, diarrhœa with or without abdominal cramps, with or without thyroid enlargement—the possibility of thyrotoxicosis should be thought of and careful studies made to establish or rule out its presence.

The most valuable single corroborative laboratory test when positive is the presence of an increased basal metabolic rate. However, in atypical or borderline cases of thyrotoxicosis the basal metabolic rate may be only slightly raised or normal.

In doubtful cases the iodin therapeutic-diagnostic test, with marked amelioration or aggravation of symptoms after iodin medication, may be helpful in determining the presence of thyrotoxicosis.

Similarly, the radium therapeutic-diagnostic test helps in atypical or borderline cases to differentiate between thyrotoxicosis and pure neurosis. Marked clinical improvement following radium application over the thyroid speaks in favour of an underlying thyrotoxicosis.

The radium therapeutic-diagnostic test properly carried out by an experienced radium therapist is a safe and dependable method, yielding the desired information without subjecting the patient to the dangers of operation and its frequent sequelæ, e.g. myxœdema.

R. G. G.

PSYCHOSES

[98] **Crime in schizophrenic types.**—James Hunter Murdoch. *Jour. of Ment. Sci.*, 1933, **79**, 286.

From his observations on criminal statistics, the author notes that crime in schizophrenics is found most frequently at those times when crimes are most

frequent. Criminal schizophrenia occurs most frequently at that age at which most crimes are committed. Between the ages of 16 and 21 recognizable criminal schizophrenics are not so common as such: (*a*) some have probably not exhibited their underlying state of mind, and are in prison or Borstal; (*b*) some are in mental hospitals; (*c*) some have not yet become insane at all. The discharge-rate of schizophrenia compares favourably with that of the average mental hospital. The prolongation of the period of care and control renders the recoveries more likely to be permanent. Heredity does not adversely affect the discharge rate. Previous attacks of insanity impair the prospects of recovery. The earlier the care and control and the longer the period of treatment, the better are the prospects of recovery.

C. S. R.

[99] **Schizophrenia in children.**—Howard W. Potter. *Amer. Jour. Psychiat.*, 1933, **12**, 1253.

From this study it appears that a typical schizophrenic reaction may put in its appearance long before the initiation of pubescence. Because the child is limited in his verbalizations and his thinking is in the direction of concreteness rather than abstraction what little delusional formation there is is simple and naïve. The outstanding symptomatology is found in the field of behaviour and a consistent lack of emotional rapport. The drive for integration with the environment, so characteristic of normal children and so essential for their personality development, is outstandingly absent. The six cases reported and discussed here represent typical schizophrenia in children. Their actual ages on admission were 4, 6, 10, 11, 12 years respectively. The three patients who were over 10 on admission presented undoubted evidences of the existence of their schizophrenia long before that age. With the exception of one case, none showed any physical indication of puberty. None of the cases showed any marked improvement over the period of hospitalization, ranging from three to 18 months. The treatment was that of continuous effort to intrigue the child's interest through a carefully organized daily routine, modified to fit the individual. It was not possible to do a great deal with a direct psychotherapeutic approach because of the lack of an essential emotional rapport. There is a superficial resemblance of schizophrenic children to certain unstable mental defectives. The schizophrenic child often appears mentally deficient because the libido is invested within the patient himself, thus interfering with the objectification of the intellectual processes. The writer thinks that a careful psychiatric study, from a psychodynamic approach, of the patients in institutions for mental defectives, might demonstrate that schizophrenia in children is not as rare as is now generally believed.

C. S. R.

[100] **Bromide delirium and other bromide psychoses.**—MAX LEVIN. *Amer. Jour. Psychiat.*, 1933, **12**, 1125.

SEVEN cases of bromide delirium and one case of bromide hallucinosis are presented. In addition to the commonly known symptoms of delirium, the patients in this group showed to a noteworthy extent two disturbances: (1) disturbances of sensation (referring not to hallucinations, but to subjective difficulty in recognizing colours, macropsia, micropsia, etc.); (2) disturbances of the association of ideas of the type commonly seen in schizophrenia. It is suggested that these two are amongst the 'ingredients' of disorientation. The delirious individual misidentifies objects and situations partly because he does not perceive well and partly because, owing to a faulty association of ideas, he discriminates improperly what he has perceived. In addition to simple bromide intoxication, delirium and hallucinosis are clinical types. There seems to be a possibility that bromide intoxication may also manifest itself as a paranoid state. No case has been reported in which a paranoid state supervened during bromide intoxication without a previous delirium. The distinction between paranoid and delirious disorientation is demonstrated.

The possibility of post-intoxication psychosis is suggested by a case in which a woman previously non-psychotic developed a bromide delirium which, four weeks after the discontinuance of bromides, merged into a chronic delirium with marked psychomotor embarrassment. It seems possible that under certain conditions bromide intoxication may precipitate a chronic psychosis which remains long after the intoxication has subsided. The etiological factors of bromide psychoses are reviewed, and attention is drawn to several objections to the recently proposed hypothesis of the occurrence of 'withdrawal delirium.'

C. S. R.

[101] **Changes in the clinical signs and laboratory findings in various types of psychoses under the influence of subcutaneous administration of oxygen.**—J. NOTKIN, J. G. WILLIAM GREEFF, F. H. PIKE, and J. A. KILLIAN. *Amer. Jour. Psychiat.*, 1933, **12**, 1271.

THE initial findings for all groups showed a definitely normal hæmoglobin count in all but a few cases which were low. There was no significant or general change after the course of oxygen therapy. This was correlated with a normal or low oxygen content, very few high values being observed. Both readings were in general slightly increased, if there was any change at all, in involution melancholia, epilepsy and dementia præcox. The manics showed a slight decrease in both. No striking variations were observed in the erythrocyte counts, and in the majority of cases the leucocyte count was high before oxygen and treatment seemed to aggravate the condition. All groups had normal or low readings for polymorphonuclear counts, no high counts being noted except in dementia præcox. The lymphocytes were

normal throughout with a lack of low readings in the dementia and epileptic groups. All cases had normal basophils, endothelial cells, and eosinophil counts throughout the observation period, except for a small percentage of high values in the eosinophils of the dementia præcox group. Thrombocytes had some low readings in the dementia præcox and melancholia cases. There was nothing characteristic about the blood-pressure, but the pulse-rate decreased in all cases except for the manic group, in which some increase was observed. The basal metabolic rates were mostly normal. High values were found in the groups of epileptics and melancholias. The oxygen content was low in most cases. In all groups except the melancholias there was some improvement of this condition on treatment. Oxygen therapy produced no consistent results, except perhaps that the improvement of dementia præcox and manics was more pronounced than the increase in the other two groups. The improvement in the catatonics may be ascribed to the improved capillary circulation, evidenced by the decrease in the cyanosis of the extremities.

C. S. R.

[102] **Depressive reaction types.**—W. McC. Harrowes. *Jour. of Ment. Sci.*, 1933, **79**, 235.

After making a survey of the many different views held on the subject, the writer concludes that the affective reaction-patterns of the depressive type should be regarded as being an attempt on the part of the patient to manage his social and personal integration by means of a regulative mechanism which is not functioning economically. Recapitulation of the psychobiological development of the individual makes it abundantly clear that affect as a regulator exercises a progressively more delicate type of control as age advances. It would seem that in the formulation of broad depressive groups there appears a set of alternative, side-track types of behaviour, equivalent experiments to normal affect mechanism, but not capable of being brought to useful conclusions. During the clinical examination of depressives and their social setting, it becomes well established that we are dealing with circumstances where specific affective responses have been much more unhelpful than otherwise, usually demonstrably so over a long period of time—unlike the state of affairs in the better integrated, where emotional drives help to push the individual to some end-result of satisfaction. Basically the specific emotional 'rises' of the depressive types work in terms of inhibition, either predominatingly psychological or biological. The extent to which motivation comes in is less important, because behind the motivation is the psychobiological constitution of the individual. Experience with depressive cases sooner or later leads to an appreciation of, and respect for, the biological features of a condition where, as Benon says, the best psychotherapy is sympathetic discussion 'sans métaphysique.'

C. S. R.

[103] **Notes on psychogenic depression and melancholia.**—PAUL SCHILDER. *Psychoanalytic Review*, 1933, **20**, 10.

FOLLOWING the analysis of a case of prolonged psychogenic depression, the writer concludes that in such a condition the chief point of fixation is in the œdipus region and accessory fixation points in oral sadism. In melancholia there are the same points of fixation, but the fixation in the region of the œdipus complex is less important. Oral sadistic fixation is in the foreground. Generally speaking, in every neurosis and psychosis we must consider not only one point of fixation but the whole development, and there will always be accessory points of fixation in this development. The various symptomatologies of the neurosis and psychosis depend upon the primary as well as the secondary points of fixation. We may ask the question why hysteria takes the form of a depression and why it has this particular symptomatology. The preliminary answer would be that in psychogenic depression we deal not only with the primary point of fixation in the region of the œdipus complex but also with secondary fixation points in the oral sadistic sphere. The similarity with melancholia is based on a partial identity of the points of fixation, but the relative value of these fixations to the whole development and to sexual structure is different.

C. S. R.

[104] **Bulbocapnine catalepsy in man** (La catalepsie de la bulbocapnine chez l'homme).—M. ELLERMANN. *Acta Psychiat. et Neurol.*, 1933, **8**, 347.

AFTER citing the experience of other investigators, the writer briefly recounts his own work with 15 female patients, of whom all except one were schizophrenics, the other being an epileptic. Using different doses of bulbocapnine, and by different routes, he was unable to produce somnolence, flexor attitudes, psychomotor torpor, amimia, catalepsy, or even the accentuation of a previously existing state of the kind, by this method. His results were persistently negative, differing materially from those of some other observers. The conclusion is that seemingly much heavier doses will have to be used, and these are without question ' very dangerous.'

A. B.

[105] **Blood cholesterol studies in mental disease : Schizophrenia.**—PURCELL G. SCHUBE. *Amer. Jour. Psychiat.*, 1933, **12**, 1227.

AFTER a survey of the literature on the subject and a discussion on the relation of cholesterol to metabolism, the results of a study of 55 cases of schizophrenia with a range of blood cholesterol values of 55 to 300 mg. are given. Many of the factors which might alter the blood cholesterol other than that induced by the mental disorder itself were investigated. It was

found that there is a definite tendency to a lowered blood cholesterol in schizophrenia as a group. There are cases in which the cholesterol is normal or increased, but the majority of cases show values below the lower limit of normal or in the lower limits of the normal range.

C. S. R.

[106] **The blood-urea in psychotics.**—D. N. Parfitt. *Jour. of Ment. Sci.*, 1933, **79**, 501.

High blood-urea levels in psychotics are most commonly found in the confusional group. In 30 successive cases, only 13 gave blood-urea findings that were always within normal limits. Of the 17 cases with raised levels, six gave figures between 45 and 70 mgm. per cent., but none of these suffered from nephritis, although in each case the diet was arranged and graded to reduce the level to normal and to keep it so. The remaining 11 cases suffered from chronic nephritis. Five of these had a blood-urea between 100 and 200 mgm. per cent., and six between 60 and 100 mgm. per cent., while confirmatory signs were found in all.

In 15 successive cases of mania 11 had normal blood-ureas. The remaining four had raised levels above 60 mgm. per cent., and three others, without evidence of chronic nephritis, were all complicated by a degree of confusion which improved rapidly on a strict milk and water diet. Blood-urea estimations in three cases of acute delirious mania suggested that the results were useful guides in prognosis. Originally the figures were high and remained so in the fatal cases, but fell to normal in one that recovered. High blood-urea levels were rare in 40 cases of melancholia. In five it was 40 mgm. per cent., and only one case had a persistently high reading. In nine cases of schizophrenia the blood-urea varied between 34 and 18 mgm. per cent. Two further cases gave abnormal results. In 11 cases of non-systematized delusional insanity the reading varied between 21 and 48 mgm. per cent. In nine senile cases the results varied between 25 and 50 mgm. per cent. The blood-urea was rarely below 40 in those with high blood-pressure, even if albumen was regularly absent from the urine. Normal readings were obtained in several cases of postencephalitic psychosis and in mental defectives.

C. S. R.

PSYCHOPATHOLOGY

[107] **The thyroid gland in mental deficiency.**—J. L. Newman. *Jour. of Ment. Sci.*, 1933, **79**, 464.

The variations in the appearances of the gland are extraordinarily wide. No constant findings could be established to connect mental deficiency with histological abnormality of the thyroid. It was not possible to find any

feature which would distinguish the primary from the secondary amentias, nor were the appearances of the thyroid in any of the subtypes of these groups consistent, save only in the case of the mongols. Here all that can be said is that evidence of a qualitative and quantitative lack of parenchymatous activity suggests a diminution of thyroid function in this condition. In a series of children's thyroids a type of hyperplasia is found which is not seen in the adult. In it the epithelial cells grow syncytially and infiltrate the colloid, and the process may advance to such a degree as entirely to obscure the original alveolar structure. Another feature of the gland of childhood, one to which in the past much significance has been attached, is the so-called 'cell-masses.' It seems that these masses are of no more significance in the child than in the adult, and they are therefore of no value as a histological criterion. Some increase may be found in the fibrous stroma of the glands of quite young children in whom, on clinical grounds, there has been no reason to suppose that the thyroid had been the subject of excessive functional demands. The colloid may vary greatly in its appearance and its staining reactions, being hyaline, granular or apparently mucoid, and either acid or alkaline in its staining affinities. The state of nutrition of the patient seems to have no connexion with the histological appearances of the thyroid. Fever, on the other hand, at first leads to absorption of colloid and increased activity on the part of the epithelium. But if the fever is continued overcompensation is the result, and the gland then reverts to a condition of colloid storage with diminished cellular activity.

Though an association between the thyroid and thymus has been established in many abnormalities of the former, and though the thymus tends to atrophy prematurely in the amentias, no close correlation could be found between the two glands. But the more considerable degrees of fibrosis in the thyroid were associated with absence of the thymus.

C. S. R.

[108] **Nature of feeble-mindedness.**—ABRAHAM MYERSON. *Amer. Jour. Psychiat.*, 1933, **12**, 1205.

MYERSON offers as a working hypothesis for psychiatry and for biological medicine the concept that the most practical field is the influence of environment upon germ-plasm and upon the mind and the personality of the human being. This environment needs to be studied in its minutest phases; uterine conditions ought to be a field for research, and the vaginal smear of Stockard and Papanicoloau offers at least a hint as to how this research can be conducted. A record of intrauterine conditions is possible; at least a more careful study of such conditions can be undertaken. Within this environment the earliest and most profound influences are exerted upon the developing individual, and yet almost no research has been carried out in this field. A careful study of nutrition, before pregnancy, of the effects of

infectious diseases upon ovulation needs to be instituted. It is possible that a defective individual occurring in a normal family may represent not a hidden Mendelian character of hypothetical origin, but the effect of som passing infection upon germ-plasm. The influence of the crowding of slum under-nutrition, infectious diseases, work in factories, and poor hygien conditions is a more profitable study, according to this working hypothesis than studies of defective communities existing under traditions and actua physical environments which might in themselves explain in some measur their defectiveness. While alcohol and syphilis may not be the blasto-phthoric forces which injure germ-plasm, the case certainly is not closed against their effects, and we need not confine ourselves to the mere physic conditions which surround groups of the population in an effort to disco the blastophthoric forces. The day is past when emotional states can b dismissed as merely mental. We know that a great emotion is a bodily event reaching through every tissue and changing the chemistry of the entire organism in some measure. In this field, as in every field in which the living creature is studied, the sum total of the forces operating on the human being must be taken into account. Heredity is the name given to an inferior force or set of forces, which operates fairly stably but is constantly influenced by an immense number of other forces. To explain the defective by this inner force alone is to leave out of account the rest of life in all its varied and potent aspects.

C. S. R.

[109] **The mongol : A new explanation.**—R. M. Clark. *Jour. of Ment. Sci.*, 1933, **79**, 328.

The writer's contention is that mongolism is the aftermath of fœtal Graves' disease. The etiology and origin of Graves' disease and mongolism are the same. There is evidence to show that both diseases twin as other germinal diseases, proving the germinal origin of both diseases. There is also support for the view that the factor in the germ responsible for both diseases is a hereditary factor. A number of observers hold that mongolism is a hereditary disease, but this cannot be said to be the generally accepted view. The sterility of the mongol at first sight appears to negative heredity, but recent proof of the germinal origin of the disease and the possibility of its being the intrauterine form of exophthalmic goitre require reexamination of the question. When mongolism occurs in twins it always presents itself in both twins, if the twins are uniovular, but in one only if biovular. This is strong proof that the germinal defect is the essential and major factor in the etiology of mongolism. The hereditary factor may be a unit character, it may be connected with the endocrine system, the periphery, the central or autonomic nervous system. Such a factor seems more likely to bring about a definite

and familial disease, such as exophthalmic goitre, than the imaginary 'neuropathic' or 'morbid' hereditary factor often suggested. It is thought that the facts presented seem to justify the following deductions: Exophthalmic goitre may occur in utero, just as it does in after life, for the necessary hereditary factor and environmental conditions are at times both present in utero. When exophthalmic goitre occurs in utero in twins, it will present itself in uniovular and biovular twins in the same way as mongolism; in both exophthalmic goitre and mongolism a susceptibility is inherited; the determining factors in both diseases seem to be the same environmental conditions. In mongolism the environmental condition ceases at birth.

C. S. R.

[110] **The microglia in mongolian idiocy** (La microglia nell'idiozia mongoloide). —F. CARDONA. *Riv. di pat. nerv. e ment.*, 1933, **41**, 293.

THE brains of two typical cases of mongolism were examined for microglia. Only a slight numerical increase in microglia was noticed, but there were no signs of serious progressive or regressive lesions of it. At the base of the brain it was normal. This confirms the hypothesis that the lesion in mongolian idiocy is mainly cortical, which region shows a constant hypoplasia. It is against the theory that the chief lesion is in the basal ganglia and that inflammatory lesions are present, suggestive of syphilis.

R. G. G.

[111] **The human-figure drawings of adult defectives.**—C. J. C. EARL. *Jour. of Ment. Sci.*, 1933, **79**, 305.

IN a study of the drawings of 420 adult mental defectives, the writer reaches the following conclusions. The Goodenough drawing test is a useful component for a performance battery for these patients. In a group of 113 patients whose mental ages lie between five and nine years, the coefficient of correlation between this test and the Stanford-Binet is $+ \cdot 48 + \cdot 07$. Compared with normal children of similar intelligence, defectives excel in representation of detail, but are markedly deficient in correctness of proportion, and of spatial orientation; their drawings display a certain 'incoherence.' Defectives' drawings are of static type, and tend towards femininity. A special ability for drawing is uncommon in defectives. The ability is more apparent than real, and true artistic talent does not occur. The drawings of psychotic and unstable subjects frequently show distinctive features, notably perseveration, idiosignificant detail and marked sex inversion.

C. S. R.

[112] **The disintegration of nervous tissue into the cerebrospinal fluid in mental illnesses** (La disintegrazione nervosa nel liquor in rapporto alle malattie mentali).—O. Meco. *Riv. di pat. nerv. e ment.*, 1933, **41**, 619.

The author holds that in certain mental conditions it is possible to demonstrate, by a special silver solution causing a precipitation in the cerebrospinal fluid, products of disintegration of the central nervous system. Thus his tests were positive in status epilepticus, in malarial attacks during the treatment of general paralysis, in senile dementia, in excited or confusional states, in catatonic stupors and in poisoning by luminal, while they were negative in other mental conditions.

R. G. G.

[113] **Experimental toxic approach to mental diseases.**—A. Ferraro and J. E. Kilman. *Psychiat. Quarterly*, 1933, **7**, 115.

The authors have studied experimental intoxications in animals produced by indol and histamine chiefly—poisons normally present in the intestinal tract. Pronounced pathological reactions were found in the brain, and these are described at length. The experimenters also show that when the protective mechanism of liver, lungs, and intestines is broken down quite small amounts of indol are injurious to the central nervous system. The same finding applies to histamine. Further, association of the two in morbid activity undoubtedly shortens the duration of the animal's life and aggravates the changes found in brain tissue.

The important aspect of this contribution, however, deals rather with the application of the data to the question of pathogenesis in mental disease. Pathological changes occurring in the intestinal mucosa must reduce the protective action of that organ in detoxicating indol, and a similar remark is applicable to the liver. Various psychoses are known clinically to be preceded or accompanied by gastrointestinal autointoxication ; the authors claim that their experiments give a lead to the placing on a sound foundation of the view that endogenous poisons are in some cases etiologically responsible for mental symptoms.

J. V.

[114] **The histopathology of the cerebral hypophysis in general paralysis** (Contributo alla conoscenza istopatologica dell' ipofisi cerebrale nella paralisi progressiva).—G. L. Francesconi. *Riv. di pat. nerv. e ment.*, 1933, **41**, 661.

Twelve hypophyses from cases of general paralysis were examined with the following results :—

1. A definite inflammatory reaction of the lymphocytic type, both in the tissues and vessels in the capsules and in the connective tissue septa, was found.

2. A severe degenerative reaction, especially in the anterior lobe, was seen in almost all cases.

3. Definitely inflammatory reactions in the tissue of the gland itself, with lymphatic infiltration, occurred.

4. A relative quantity of iron pigment in the posterior lobe was remarked in almost all cases.

The author claims that these changes in the hypophysis in general paralysis are constant and characteristic.

R. G. G.

PROGNOSIS AND TREATMENT

[115] **The difference between Freud and myself** (Cio che mi separa da Freud).—W. STEKEL. *Arch. gen. di neurol. psichiat. e psicoanalis.*, 1931, **12**, 8.

STEKEL explains the doctrine and practical differences between his own teaching and that of Freud. He denies the necessity of the theory of the libido and the original traumatism as a basis of a psychoneurosis, but affirms that every case is based on a psychic conflict which ought to be investigated without preconceived methods or precedent dogmas. Stekel prefers an active method of treatment to the purely passive method of Freud, and holds that this will depend on the intuitive ability of the analyst to discover and remove the psychic blind spots of the patient, which prevent his adapting himself to his family and the world. Stekel does not admit the depth of the unconscious which Freud insists on and accepts interpretations differing entirely from the Freudian concepts. He denies the importance and frequency of the Freudian complex. He holds that treatment should last as short a time as possible, probably not more than four months. He admits the invaluable service rendered by Freud to psychological medicine, but regards his own methods as more practical and promising.

R. G. G.

[116] **The investigation of a specific amnesia.**—MILTON H. ERICKSON. *Brit. Jour. Med. Psychol.*, 1933, **13**, 143.

THE problem investigated was an attempt to recover the content of a specific amnesia without the aid of supplementary information. Techniques of free association alone, free association and direct questioning in hypnosis, automatic writing, and crystal-gazing were tried without success. Finally, by means of a specious argument concerning the existence of a third level of

consciousness and the permission to use disguise mechanisms, the forgotten material was actually obtained, but in such guise that its true significance was not recognized. Then by means of dream activity a situation was created whereby the subject, without assuming the responsibility, could circumvent the repression. Following this a conflict state developed, characterized by the alternate repression of two ideas, and ultimately resolved by the attachment of the affect to a related subject, from which it was eventually dispelled. The motivation of the amnesia was not learnt until much later, when the subject disclosed its origin to lie in an emotional conflict concerning two men.

C. S. R.

[117] **Some experiments in reinforcing mental analysis in cases of psychosis.**—CLIFFORD ALLEN. *Brit. Jour. Med. Psychol.*, 1933, **13**, 151.

AUTOSUGGESTION in psychoses has been tried unsuccessfully in the past and abandoned as useless. The experiments here related seem to show that the cause of the failure is that the suggestions have been wrongly applied and have been directed to increasing the repressions on some powerful complex. The experiments of the author indicate how suggestions can be applied to influence complexes with safety and some certainty. Mental analysis can thus be reinforced with definite results. Four illustrative cases are given.

C. S. R.

[118] **On the interruption of the catatonic syndrome** (Sulla interruzione della sindrome catatonica).—S. GULLOTTA. *Riv. di pat. nerv. e ment.*, 1932, **40**, 241.

CATATONIAS can be interrupted by the administration of narcotics, and the use of the barbiturate series is the best in view of the constancy of the results and absence of ill effects. Twelve catatonics were given sodium amytal intravenously with uniformly good results so far as arousing the patient from the catatonic condition is concerned. When so aroused, a psychological examination is possible, which may determine a diagnosis otherwise difficult to establish. The author concludes that since sleep interrupts catatonia the latter is due to a physiological disturbance of the central nervous system, probably situated in the subcortical region, and to an absence of cortical inhibitory influences. When catatonia ceases this is probably brought about by a restoration of balance between these subcortical centres and the controlling cortex.

R. G. G.

[119] **On the action of barbituric acid compounds : A contribution to the prolonged narcosis treatment of mental symptoms.**—A. M. MEERLOO. *Jour. of Ment. Sci.*, 1933, **79**, 336.

SUFFICIENT clinical material regarding barbital intoxication has been collected to make it possible to draw conclusions, while sifting the complications, in regard to the action, point of contact and so forth of barbitals. Within the limits of psychopathology we must consider this sedative as a specific poison, which induces and affects local biological processes, but at the same time as a non-specific factor, which only lays bare a more deeply buried psychic structure. Clinically speaking, we may resolve the action of barbitals to action on the subcortical brain centres—the brainstem in its widest sense. Pathologicoanatomical discoveries support this. The secondary phenomena produced by barbitals, such as catatonia, epilepsy, hallucinations, etc., are of interest to psychopathology ; they constitute a contribution to our knowledge of the function of the palæencephalon. Prolonged treatment of psychotic patients with barbitals is of great therapeutic value. The effect of this may be compared with that of nonspecific stimulant therapy. Sudden disintoxication is an important factor here, and sleep constitutes no very real element. This therapeutic method—especially as far as concerns the avoidance of dangerous secondary action—is susceptible of improvement, and needs further systematic study.

C. S. R.

[120] **A note on the use of nephritin in psychotics.**—D. N. PARFITT. *Jour. of Ment. Sci.*, 1933, **79**, 371.

THE action of the internal secretions of the kidney is a debatable subject, but some illustrative cases given seem to show that nephritin has a definite if limited use in certain psychotic cases with renal insufficiency. Its use would seem to be confined, at least in chronic nephritic conditions, to those cases with azotæmia which fail to respond to stringent dietary restrictions ; in these, with an adequate dosage of at least 20 tablets a day, the improvement is often remarkable. The preliminary and concurrent limitation of diet to milk and water in equal parts, with glucose and fruit-juice, is essential. The mental improvement is proportional to the degree of confusion present due to renal toxæmia, and where this is absent no change is produced in the psychosis. If dieting alone produces a marked improvement, then nephritin will do no further good even if the renal condition is still unsatisfactory. The azotæmia of wasting conditions is unaffected by nephritin.

C. S. R.

Reviews and Notices of Books.

Human Mentality in the Light of Psychiatric Experience. By Bror Gadelius, Stockholm. Copenhagen : Levin and Munksgaard ; London : Humphrey Milford. 1933. Pp. 620. Price 30s.

THE volume under review is an English translation of the first two parts of the second edition of Professor Gadelius' textbook, published in Swedish. It represents the prolegomena of that work, the general section of an outline of psychiatry, and deals with many matters of perennial interest for the student of the subject. After an opening chapter, historical in cast, giving an account of the care of the insane through the centuries, the main division discusses the functional structure of mental life and its morbid changes in a series of topics whose nature is sufficiently indicated by some of the titles, e.g. ' The Morbid Changes of Emotional Life,' ' The Psychopathology of Thinking,' ' Disorders of Memory,' ' Perversions of Instincts,' and such like. In the second division the causes, and in the third the treatment, of mental disease are examined.

What strikes the reader of this admirable exposition of present-day psychiatric doctrine is the breadth of view of its author, his philosophical and critical spirit, his caution in reaching conclusions, and, perhaps most of all, his appreciation of the importance of embryology, neurology, and endocrinology where psychiatric problems are concerned. It is gratifying to note his definite statement that the midbrain, vegetative centres, and other parts of the nervous system ' are of far greater interest to psychology as well as to psychiatry than was surmised a few decades ago.' Indeed, he avers that ' the most important gain of psychiatry during the last twenty years' is precisely the connexion of the endogenous insanities with, inter alia, the midbrain.

Of especial interest also, from another standpoint, is the chapter devoted to psychoanalysis as a theory and as a mode of treatment. Here will be found a cogently argued criticism of Freudian doctrines, in which his view of the value of Freudian theory and practice is clearly and pointedly exposed. He lays stress on its ' irremediable weaknesses,' its ' extreme onesidedness,' its ' serious errors,' its ' degradation of consciousness,' and numerous other aspects also come under his critical condemnation. He particularly underlines the undue universality assigned the Œdipus-complex, and cites the comment of Hoffding, that Freud has ' indecorously debased ' Sophocles' great drama, where the tragic development rests on ignorance of the close relationship between son and mother. So manifold are the topics discussed in this chapter that they cannot even be summarized within the limits of our space, but the criticisms of Freudian dream interpretation must be specially noted ; Professor Gadelius shows that the mode of reasoning adopted by the

psychoanalyst in this regard 'may as easily prove the existence of an Œdipus-complex as the thesis that the moon is a green cheese.' 'He arrives where he wishes to arrive'; 'the evidence for the results arrived at in Freud's *Traumdeutung* is entirely valueless because it is not unprejudiced.' Professor Gadelius quotes dryly the assertion of the Freudian Dr. Sadger: 'If a person otherwise endowed with common sense and a normal intelligence does not understand the least of the secrets of mental life unveiled by Freud, he is either a professional psychologist or a psychiatrist.' Such echoes of unfruitful controversy, however, need not be taken too seriously; what must be pondered over by the open-minded reader is the whole trend of the chapter, for it certainly constitutes one of the most well-informed critiques of recent years.

Neuropathology. By Walter Freeman, M.D., etc., Professor of Neurology, George Washington University, D.C. With 116 illustrations. London and Philadelphia: W. B. Saunders Co. 1933. Pp. 349. Price 20s. net.

Professor Freeman has compiled a serviceable compendium of neuropathology, well written and well illustrated. Its outlook is essentially modern and it ranges over a wide field. A preliminary chapter on cytology and cytopathology is followed by sections devoted to various types of pathological process (inflammations, intoxications, injuries, malformations, degenerations) or concerned with topographic changes (the meninges and their diseases, vascular disease) or with specific morbid agents (tuberculosis, syphilis). There is thus a cross-classification which might have been obviated with advantage. In some respects, also, the allotment of space to this or that affection leaves something to be desired; to spinal tumours three and a half lines are devoted; the descriptions of syringomyelia and amyotrophic lateral sclerosis (notably the latter) omit various points which have a cardinal bearing on the nature of the conditions; amyotonia congenita is annotated without reference to the Werdnig-Hoffmann type in relation therewith. Among brain tumours it is stated that papilloma of the choroid plexus does not metastasize, but this is doubtless a mere slip, though it needs correction. We are told that myoclonus epilepsy is 'one of the few nervous diseases in which specific changes occur in ganglion cells,' yet various observers have failed to discover the amyloid inclusions or bodies which Professor Freeman describes. So vague or at least uncertain, however, are pathological data in respect of a number of nervous affections that it would be unfair to expect critical discussion in a book of this kind; as far as it takes the reader, it can be recommended for its concise and, on the whole, adequate presentations.

Of the glossary which is appended we can only say that a large number of the words placed therein require no explanation, while other definitions themselves are in need of this, e.g. 'Oligodendroglia—the element of importance in myelinogenesis and possibly in myeloclasis.' Still other terms are ignored (e.g., collagen, reticulo-endothelial system, fibroglia).

The Mechanism of Nervous Action. By E. D. Adrian, M.D., F.R.S., Foulerton Research Professor of the Royal Society. London: Humphrey Milford. 1932. Pp. 103. Price 10s. 6d.

THE clinical neurologist is sure to learn very much of direct interest to him by close perusal of Professor Adrian's latest studies on the neurone. It seems invidious to single out any one of the five main topics of the book, nevertheless the chapter on pain, brief though it be (as, indeed, all are) is deeply instructive.

Professor Adrian's chief problem is that of the specific effects of different sensory fibres. If the skin is compressed we feel first touch, then pressure, then pain; are such changes in the qualities of sensation due to increases in frequency of discharge in each nerve-fibre, or do they represent the coming into action of specific varieties of nerve-apparatus? His conclusion is, that, on the whole, it is unlikely that pain is always due to stimulation of specific pain-fibres, that non-medullated fibres whose conduction is slow and whose endings respond only to noxious stimuli can convey little or nothing else, that the most sensitive and rapidly conducting tactile fibres can not, but that, 'for all we know,' intermediate medullated fibres may give either a sense of contact only, or of pain, according to the magnitude of the discharge. He does not consider there is any clear-cut division between the sizes of sensory fibre, to correspond to muscle sense, touch, and temperature; yet there seem to be distinct differences in potential and conduction rates. The tentative decision is that 'with certain fibres a change in the discharge frequency may alter the quality as well as the intensity of sensation.'

Psychoanalysis and its Derivatives. By H. Crichton Miller, M.D., etc., Hon. Director of the Institute of Medical Psychology. London: Thornton Butterworth Ltd. 1933. Pp. 256. Price 2s. 6d.

THE medical profession is indebted to Dr. Crichton Miller for more than one valuable addition to the stock of contemporary psychological and psychotherapeutic literature, and must again owe acknowledgment to him for a dispassionate examination of psychoanalysis and the movements which have originated therefrom, in small compass, relatively untechnical language, and an engaging style. The book ostensibly can be 'put into the hands of the layman,' since some acquaintance with the nature and content of psychoanalysis, we are told by Dr. Miller, has become 'an essential part of general education.' Whether this be so or not need not here be adverted to; the point is rather that the medical profession can peruse this unpretentious little volume with great benefit. It clears the air, and at the same time, we think, reveals where lies, at least to some extent, Dr. Miller's own inclination. In his preface he speaks of his adoption of an eclectic attitude, but it seems also that while he is not satisfied with any one psychological and psychotherapeutic system he yet favours one which stresses the genetic factors in the 'psychosomatic unity' and can thus lay claim to a completeness vouchsafed to none

of the rest. In another place he draws attention, among the failings of psychoanalysis, to the fact that it is isolated, that its silence on constitutional factors is striking, that it has no contacts with other branches of contemporary allied science. Endocrine correlations are simply out of the picture, and the advances of recent time in biochemistry, vegetative neurology, and the like, in which psychiatry is patently much interested, mean nothing to psychoanalysis. Reading between the lines, he who follows Dr. Miller's scrupulously fair, well-informed, and by no means unsympathetic narration of the facts and fancies of psychoanalysis, may gain a hint as to what the author thinks its future may turn out to be.

Diseases of the Nervous System. By W. Russell Brain, M.D., F.R.C.P., Assistant Physician to the London Hospital, etc. London: Oxford University Press; Humphrey Milford. 1933. Pp. 899. Price 27s. 6d.

Of recent years, more especially in America and on the Continent, various textbooks on nervous diseases have been published, either relatively condensed, or encyclopædic in their range. Among those of moderate size the one which Dr. Russell Brain has written will take a prominent place, without doubt, in view of its combination of precise and concise description with comprehensive covering of the field. His limits indeed extend beyond neurology proper, including, as they do, inter alia, Paget's disease and sclerodermia. Each condition with which Dr. Brain deals is discussed along conventional lines of symptomatology, pathology, etiology, diagnosis and treatment—and all this is done in an eminently practical fashion. The latest developments in the treatment of epilepsy, for example, are fully set forth. Initial sections are devoted to anatomy and physiology in their bearing on the symptoms of nervous disease; some disorders of function are briefly examined, others at length. In his paragraphs on aphasia the writer follows the divisions recommended by Head, although he is aware of their unsatisfactory nature in some respects. Considerable attention is assigned to the cerebrospinal fluid from its clinical aspects, to ventriculography and other present-day refinements in diagnosis.

Like other textbook-makers, Dr. Brain rather sidesteps the problems set by nosology when the nervous system is involved. His classification is sometimes anatomical ('The Cranial Nerves,' 'Disorders of the Spinal Cord,' etc.), sometimes pathological ('Intracranial Tumour,' etc.), sometimes etiological ('Intoxications and Deficiency Disorders'), sometimes clinical ('Paroxysmal and Convulsive Disorders'). This utilitarian method has its advantages when we are still ignorant of much of the causation of nervous maladies, nevertheless knowledge is progressing, and such ad hoc procedures are really obsolete. They result in a series of anomalous placings which it would have been well to avoid. For instance, spina bifida is allotted to disorders of the spinal cord and not to 'Congenital and Degenerative Disorders'; degenerative disorders, again, are distinguished from infections,

although toxidegeneration is one of the recognized forms of pathological process ; myelitis is treated in the spinal cord section and not among infective conditions—and so forth.

Some 50 illustrations are furnished, and there is at the end of the book a useful list of bibliographical references.

Pituitary Body, Hypothalamus, and Parasympathetic Nervous System. By Harvey Cushing, Professor of Surgery (Emeritus), Harvard University. With 97 illustrations. London : Baillière, Tindall and Cox. 1933. Pp. 234. Price 29s.

In this slim volume are conveniently collected four papers by Professor Cushing which have already been published but are now amplified. The first, the Lister Memorial Lecture (1930), deals with neurohypophyseal mechanisms from a clinical standpoint, and offers a closely reasoned argument that diencephalon and pituitary body are mutually interdependent—an old idea, as the writer shows, now more fully substantiated. The symptomatology of craniopharyngiomas is relied on for material support. The second (1931) is concerned with various aspects of the actions and reactions associated with pituitrin (posterior pituitary extract), especially those which occur when the active principle enters the cerebrospinal fluid and thus may come to act on diencephalic nuclei of parasympathetic class, producing salivation, lachrymation, sweating, flushing, vomiting and hypothermia. As for the third, it is possibly the most impressive and informative, dealing as it does with the question of pituitary basophilism at length, and in an illuminating way. Professor Cushing faces the problem of the appearance of supposedly adrenal phenomena in clinical cases shown to have basophil adenomas of the pituitary, pointing out that such phenomena also characterize some examples of gonadal or pineal affection, but concluding that in all future instances of the syndrome the anterior pituitary must be searched minutely for basophil manifestations. The fourth (1931) discusses the peculiar interconnexion of peptic ulcer and the interbrain, and concludes that a parasympathetic centre, which can be influenced by cortical or psychical stimuli, is present in the diencephalon. In this way peptic ulcer may be of neurogenic origin.

Paralysis in Children. By R. G. Gordon, M.D., D.Sc., Physician, Bath and Wessex Orthopædic Hospital, etc., and M. Forrester Brown, M.D., M.S., Surgeon, Bath and Wessex Orthopædic Hospital, etc. London : Oxford University Press. 1933. Pp. 328. Price 15s.

This is an encouraging book, and for two distinct reasons. In the first place it represents that intimate collaboration between neurologist and orthopædist which is patently desirable but often lacking, and its whole theme is that the treatment of paralytic syndromes is far from being so nugatory as is often

imagined. For here are photographs showing how hemiplegic and paraplegic limbs can be made to render good service despite pre-existing contractures, how poliomyelitic disabilities can be overcome, how spasticity and rigidity can be counteracted. The writers' plan is to describe in detail the restorative measures employed, and their systematic application to a considerable number of nervous affections in children, of differing etiology. Among the more interesting and important of the remedial techniques discussed a foremost place is taken by re-education in a bath of water at a temperature of 90°–100° F. The book has many good illustrations, is well written, and provided with brief bibliographies.

Incidentally, the 'Hoffmann-Werdnig' type of amyotrophy, as so written by the authors, is generally termed 'Werdnig-Hoffmann'; and the Dejerine-Sottas form of neuritis is by no means always due to congenital syphilis, as they allege.

Behaviour Mechanisms in Monkeys. By Heinrich Klüver. With an introduction by K. H. Lashley. Chicago: University of Chicago Press; Cambridge: University of Cambridge Press. 1933. Pp. 387. Price 22s.

THE latest volume in the *Behaviour Research Fund Publications* embodies the results of numerous experiments conducted on subhuman primates by Dr. Heinrich Klüver. He has studied the behaviour of both old- and new-world monkeys under a great variety of experimental conditions, with a view to ascertaining, if possible, any basic mechanisms underlying their responses. In highly ingenious fashion he has devised new techniques for eliciting behaviouristic reactions, seeking to determine how his animals adapt themselves to changes in stimuli of an auditory and visual kind, and more especially as regards those connected with the sense of weight. By changes in the external stimulus situation Dr. Klüver has been able to show that monkeys give differential responses which are not upset by environmental variations, and assumes that constancy of response in these circumstances depends on the existence of stimulus-properties rendering heterogeneous stimulus-combinations 'identifiable.' But whether the animal 'generalizes,' or 'abstracts' these stimulus-properties, is debatable.

The book is technical and its descriptions of experiments are long and detailed; its reading is, however, facilitated by convenient discussions and summaries.

Zur Biologie der Ethik. By Dr. Otto Kant, Privatdozent for Psychiatry and Neurology at the University of Tübingen. Vienna: Julius Springer. 1932. Pp. 160. Price RM 8.80.

ADOPTING a purely empirical approach to his subject, Dr. Otto Kant furnishes in small bulk a thoughtful discussion of problems arising out of the sense of

guilt. He distinguishes between two types of this feeling, real and fictitious, and indicates the insufficiency of the older conception of Nietzsche, that it was derived from weakening of the 'will to power.' This view ignores such important factors as the compelling need to surrender. In the first form of the condition, animal impulses come in conflict with the whole psyche, and are wrecked against the moral ego-ideal. A triad of components can be discovered—recognition of the existence of the animal impulses, realization that impulses, not felt positively, can nevertheless come into action by 'perverse' ways, and self-depreciation. The second variety seeks the cause of some fateful failure in an unreal, non-genuine, guilt. The bearing of these on numerous questions which interest the psychopathologist is carefully annotated, as well as on their significance for problems of ethic.

Soziologie der Neurosen : die Nervosen Störungen in ihren Beziehungen zum Gemeinschafts- und Kulturleben. By Dr. Karl Birnbaum, Professor in the University of Berlin. Berlin : Julius Springer. 1933. Pp. 87. Price RM 4.60.

DR. BIRNBAUM'S little essay on the sociology of the neuroses starts from the position that in neurosis-formation social factors are pre-eminent, though not by themselves all-sufficing for its pathogenesis. He examines in some detail the disturbance of external social relationships as a precipitant, then those of an internal or personal kind (mental conflicts arising in the psyche and having their basis in friction between egoistic and social trends and requirements). At the same time the essayist is well aware of the part played by the constitution of the individual, discussing with insight the situation of the neurotically disposed person in the social world of to-day. The types assumed by neurosis are also described ; he says much of 'want-neurosis,' arising from manifest economic, financial, nutritional needs, and attempts a statistical record of neurosis as it occurs among different social classes, pointing out how incompletely this aspect of his theme has hitherto been studied. His paragraphs on the 'social therapy' of neurosis do not contain any particularly novel suggestion, though stressing the part which the physician can play as an adviser and helper from a social viewpoint. He distinguishes a 'Milieutherapie' from a 'Charaktertherapie,' laying down useful generalizations with which, however, most social workers are familiar.

Stoke Park Monographs on Mental Deficiency and other Problems of the Human Brain and Mind. No. 1. Edited by Richard J. A. Berry, M.D., F.R.C.S., Director of Medical Services. London : Macmillan and Co. 1933. Pp. 249. Price 10s. 6d.

THE Stoke Park Colony near Bristol houses some 1,700 patients, and was the first to be certified under the Act of 1913 as an institution for mental defectives. Well staffed and well equipped, under the enthusiastic leadership of Professor

Berry it has become the centre of good research work on mental deficiency problems, some studies of which are printed in this Monograph—17 in all. They deal with diverse aspects of the general subject—clinical, pathological, psychological, etiological—as well as with cognate questions, e.g. brain structure in relation to the mind, heredity in mental deficiency, the correlation of size of head and intelligence. A number of the papers have been published already (one in the pages of this JOURNAL). Together they form a valuable contribution, auguring well for future studies from the same source.

Anatomie Medico-chirurgical du Systeme Nerveux Vegetatif (Sympathique et Parasympathique). By J. Delmas and G. Laux. Paris: Masson et Cie. 1933. Pp. 266. Price 100 fr.

THERE is still room for monographs on the anatomy of separate parts of the nervous system, and this on the sympathetic and parasympathetic systems is very welcome. Professor Duval claims in the preface that this is the first time that the sympathetic and parasympathetic systems have been considered together in an anatomical treatise, and such a survey has obvious value. In addition to describing fully the peripheral ganglia and plexuses, the authors have devoted considerable space to the central representation of these systems in the basal ganglia, hypothalamic nuclei, brainstem and spinal cord. They have aimed both at clearness and at synthesis and have certainly succeeded in their endeavour. The book is illustrated on almost every page with useful diagrams, and there are in addition fifteen full-page drawings, the last eight of which illustrate the methods of surgical approach to the sympathetic system. These illustrations are all models of clearness, and the plates, which are drawn from actual dissections, may be taken as accurate.

The terse and apposite descriptions in the letterpress and the excellence of the illustrations are typical of the French genius for anatomical treatises. The present work is a worthy successor to such classics as 'Dejerine' and 'Foix-Nicolesco,' and should have as wide a welcome among English-speaking neurologists.

Traité de Physiologie Normale et Pathologique. Tome IX, Système Nerveux (Première partie). Edited by G. H. Roger and Leon Binet. Paris: Masson et Cie. 1933. Pp. 566. Price 100 fr.

THE physiology of the nervous system cannot be studied entirely in the laboratory, for much of our knowledge has been and continues to be derived from observations of patients. For this reason the subject is probably best approached by a treatise such as this to which clinicians as well as laboratory workers contribute. This method has its difficulties, the chief of which is to find writers who combine long clinical experience with up-to-date scientific knowledge. There has also been a constant tendency for clinicians to interpret their observations according to preconceived opinions. For these reasons

certain of the chapters are inadequate on the scientific side, and are not in agreement with English teaching on the clinical side. An exception must be made in favour of the chapter on the basal ganglia, which is an excellent résumé of our knowledge of the functions of these structures. Another criticism that might be made is that much recent work from English and American laboratories has been omitted. It is true that the rapid advances in the physiology of the nervous system make it impossible for any book to be up to date, but the bibliographies contain very few references to articles which have appeared in English within the last five years.

With these reservations we welcome a book which at least attempts to give the neurologist a review of the present position of nervous physiology. When rapid advances are being made on every side it is valuable to have such statements issued from time to time, and we look forward to the tenth volume of this treatise which is to complete the neurological section.

The present volume includes chapters on 'The Neuron,' by Verne, 'Wallerian degeneration,' by Couvreur, 'The processes of disintegration of nervous tissue,' by Ivan Bertrand, 'Nerves and reflexes,' by Cardot and Laugier, a suggestive chapter on 'Tropism,' by Bohn, 'Sensation and motion,' by Lévy-Valensi, 'Cerebral localisation,' by Alajouanine and L. Cornil, 'The basal ganglia,' by Lhermitte, 'The cerebral circulation,' by Binet, 'Convulsions,' by Richet, 'Sleep,' by Legendre, and 'Anæsthesia,' by Nicloux.

The absence of an index is a real loss in a book of this kind.

J. G. G.

Emotie, Bloedsuiker, en Hersenstam. By Dr. A. L. C. Palies, Groningen. Assen: Van Gorcum and Comp. 1933. Pp. 82. Price not stated.

In his thesis for the doctorate at the University of Groningen, Dr Palies has made a useful contribution to the interrelation of body and mind by a series of experiments designed to show how bodily processes are influenced by the emotional life. He took 100 cases of heterogeneous nature, most, though not all, being neurological, and, using routine lumbar puncture as an emotional stimulus, calculated the blood sugar of every case—before and after. He also took, by way of control, an entirely different series of 100 cases and noted how blood sugar varied in them at precisely the same time and under identical conditions except for the lumbar puncture. In 77 of the first 100 an increase in blood sugar occurred, and this is attributed by Dr. Palies solely to the 'emotion' of the puncture. Exceptionally, in the remaining 23 it either remained steady, or fell. He then ascertained that no fewer than 16 of these patients were suffering from presumptive disease of the basal ganglia, and argues that lesions in the subthalamic region interfere with the action of emotion on this particular form of metabolism. He goes a step farther, indeed, in affirming that that part of the brain is where the emotional mental

life intervenes in the functions of the vegetative nervous system. Whether such inferences are permissible, however, is open to question.

Outline of the Cranial Nerves. By John Favill, M.D., etc., Associate Clinical Professor of Neurology, Rush Medical College, Chicago. Chicago: University of Chicago Press; Cambridge: Cambridge University Press. 1933. Pp. 112. Price 8s. 6d.

In convenient form Dr. Favill supplies the student with a vademecum of the cranial nerves, in which the anatomy, functions, pathology, and methods of testing for each nerve are set down succinctly. In the case of the eighth the reader is given a particularly full account of vestibular reactions and of their clinical value, supplemented by tables and diagrams. So far as we have noted this little book is singularly free from error or mistake; it certainly contains much information in small space. The diagrams are simple, accurate, and well drawn.

Mental Diseases. Catechism Series, second edition. Edinburgh: E. and S. Livingstone. 1933. Pp. 80. Price 1s. 6d.

This little primer of mental diseases, written in the question-and-answer style, meets with some adequacy the minimum requirements of those who find themselves in the position of having to sit for examination in the subject. The descriptions are simple, the definitions clear, the point of view modernized. It would not be difficult, however, to supply illustrations of the fact that some recent advances have not been incorporated, that some of the clinical accounts are too stereotyped, that some of the treatments might be improved.

The Practical Medicine Series of Year Books: Neurology and Psychiatry. Edited by Peter Bassoe, M.D., Clinical Professor of Neurology, Rush Medical College, Chicago, and Franklin G. Ebaugh, M.D., Professor of Psychiatry, University of Colorado School of Medicine. Series 1932. Chicago: The Year Book Publishers Co. 1933. Pp. 488. Price $2.25.

The year-book series of current medical literature, reviewed in abstract, is widely known for its practical usefulness and up-to-date character. The present volume, concerned with neurology and psychiatry, will bear comparison with any of its predecessors, providing as it does short accounts of scores of articles which were published during 1932. The choice made by the editors is excellent, and the abstracts are uniformly well done. Neurology takes perhaps three-quarters of the volume, and psychiatry the remaining fourth. It is a book for the library shelves of the specialist who keeps abreast of his own subject.

Arbeiten aus dem Neurologischen Institute an der Wiener Universität. Vol. XXXV. Leipzig and Vienna: Franz Deuticke. 1933. Pp. 181. Price M. 33.

THE latest volume in this well-known series contains several articles of which note should be taken. One of these is from the pen of the editor, Professor Otto Marburg, giving a full description of a typical case of basophil adenoma of the pituitary. The clinical syndrome corresponds entirely with that which may now perhaps be regarded as established, while the pathological findings confirm those of Cushing. At the same time Professor Marburg specially comments on the state of other glands (suprarenals, testis, etc.) and discusses the still unsettled question of whether the basophil adenoma per se can be held responsible for the totality of the clinical symptoms. Another paper, by Dr. Jose Gil, describes the neuropathology of a long-standing case of progressive muscular atrophy with pseudohypertrophy, occurring in a woman who died at the age of 54, and dating back for its commencement to her 'teens. Although the cells of the ventral horns were, relatively speaking, intact, with the exception perhaps of some in the mediodorsal group, those in the vicinity of the commissure and at the base of the dorsal cornua were much degenerated. A case of hemichorea, described by Dr. Tsiminakis, is shown to have been associated with contralateral softening of the cerebellar hemisphere and dentate nucleus, with secondary degeneration of the superior cerebellar peduncle and nucleus ruber. The case belongs to the class of 'Bindearm-chorea,' but the clinical account is lacking in detail, and it may be remarked that the author's description mentions 'rhythmical' twitches and movements of the affected limbs. Dr. Tronconi has examined the extrapyramidal system of nuclei and the central vegetative centres in two cases of myasthenia gravis, and concludes that so far as these are concerned the results of pathological study must be considered negative.